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LIFE TABLES FOR A SOUTHERN CHINESE FAMILY FROM 1365 TO 1849*

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INTRODUCTION

Genealogical studies of European and American families, notably by K. Pearson and A. G. Bell, have shown that such studies afford indications of the general trend of mortality and fertility of the population of which these families are a part. The probable explanation is ably given by Bell in his study of the Hyde genealogy. He states that the descendants of the seventh generation in the Hyde family owe only 1/64th of their blood to their first ancestor and 63/64ths to the new blood of persons coming into the Hyde family by marriage. Hence he concludes that the thousands of descendants of the Hyde family constitute a sample of the general population of the country rather than a sample of a particular family in which the family traits might be expected to make their appearance.

The Chinese genealogical records probably represent the only kind of available statistical data which might throw light upon the problem of vital movements of the Chinese race in the past. Modern registration of births, deaths and marriages was not instituted in China until 1914 and never has been satisfactorily carried out even in the large cities. However, the ancient tradition of keeping an account of the family lineage has been faithfully observed for many centuries throughout the country. Usually, it is the function of the family clan to take a peri-

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odical enumeration of its members and issue a new edition of its genealogical record every twenty or thirty years. In this publication, the male lineage of the family is traced from the first ancestor known, giving a personal sketch of each male, recording the name of the father, the title of social distinction, the dates of birth and death and the place of burial together with a similar description of his wife or wives including the number of sons and daughters born to each wife. As a rule, those who died unmarried under fifteen years of age are not recorded, probably because they are considered of no importance in the propagation of the family, but the records of those people over twenty years of age are probably reasonably complete due to the emphasis that is placed on family lineage in China. It seems therefore that for the adult population over twenty years of age one will find such records a valuable source of Chinese vital statistics deserving careful study.

SOURCE OF DATA

The family record from which the present study derives its data is a generous gift from Dr. Ting-an Li, a colleague of the writer in the Department of Hygiene of the Peiping Union Medical College. It belongs to a southern Chinese family in the Chung Shan district near Canton. The first ancestor came from the northern border of Kwangtung province in the latter part of the thirteenth century. Since then, the family has continued to live in this region. Probably, as is the case of most of the Chinese rural population, the chief occupation of these people has been farming. In this part of the country, the climate is subtropical in character and the land is productive. Consequently, living is relatively easier than in most other sections of China.

The first complete account in this record registers 1365 as the year of birth of the ancestor of the fourth generation. From this date, the records give a continuous family bookkeeping of nineteen more generations up to the year 1914.

COMPILATION OF STATISTICS

The process of compiling from the original record statistical data suitable for study consists of coding, extraction of information, transfer of the latter on to punch cards and finally sorting and tabulation of the punched cards forming various frequency distributions. The details which are entered on the punch card are as follows:

a — Coded number of each individual
b — Sex
c — Year of birth
d — Age at death
e — Times married
f — Number of concubines
g — Number of sons recorded
h — Number of daughters recorded
i — Number of sons married
j — Number of daughters married
k — Class of social distinction

Since the present study deals with the age distribution of mortality alone, it will be sufficient to describe how this part of the data is obtained. The age at death of each individual is determined by taking the difference in years between the year of birth and that of death as the age at death. No attempt is made to estimate age to the last birthday. This seems sufficiently accurate, as all the deaths dealt with here are of twenty years of age and over and are subsequently grouped by their age at death into frequencies of five-year intervals.

METHOD OF STUDY

To obtain the age distributions of mortality for different periods of time the individuals are first sorted according to the year of birth and then grouped into the periods 1365-1599, 1600-1699, and by half centuries from 1700-1849. Frequency distributions of the age at death of the individuals in each of these periods are then formed using five-year age groups. In this manner, a number of mortality tables are formed for different periods of time from 1365 to 1849, giving frequency distributions of deaths in quinquennial age intervals. Each table is therefore the follow through of the individuals whose births take place within a period of years and is thus in essence the d_x column of a life table, the deaths being recorded every five years instead of every year. Thus we may write

$$\sum_{x}^{\text{limit of life}} d_x = l_x$$

and

$${}_5q_x = \frac{l_x - l_{x+5}}{l_x};$$

l_x being the number of survivors at the beginning of age x and ${}_5q_x$, the probability of dying in a five year interval for each person alive at the beginning of age x. On the basis of these relationships, the mortality tables are converted into life table functions, d_x, l_x, and ${}_5q_x$. The latter form the fundamental data from which ten life tables have been constructed. They are appended in Tables 10 to 14.

Adjustment of the total deaths of both sexes for 1800-1849

Since the last family count was taken in 1914, recorded deaths of males and females born during 1800 to 1849 include all individuals who died under sixty-five years of age, but necessarily fall short of a part of those who lived to the age of sixty-five and over. This explains, as shown in Tables 1 and 2, the much lower proportion of deaths for the age interval of sixty-five and over for the period of 1800 to 1849 than for any other period. In order to make up this loss of deaths for 1800-1849, it is assumed that

$$= \frac{\text{deaths for 65 and over for 1800-1849}}{\text{deaths for 20-64 for 1800-1849}}$$

$$= \frac{\text{deaths for 65 and over for 1750-1799}}{\text{deaths for 20-64 for 1750-1799}}$$

Then, from Table 1, adjusted deaths of males of sixty-five and over for 1800 to 1849

$$= \frac{316}{710} \times 1109 = 494$$

Hence, adjusted deaths of males of twenty and over for 1800 to 1849

$$= 1109 + 494 = 1603$$

Similarly, from Table 2, adjusted deaths of females of twenty and over for 1800 to 1849

$$= 923 + \frac{418}{621} \times 923 = 1544$$

From these adjusted deaths, the ${}_5q_x$ values for the age intervals of twenty to sixty-four are accordingly adjusted for each sex. For the age intervals of sixty-five and over, the corresponding ${}_5q_x$ values for the period of 1750 to 1799 are used, since it has already been assumed that the total deaths for sixty-five and over in the two periods are proportionally distributed. These adjusted values are given in Table 15.

TABLE 1

Distribution of Observed Deaths for Males of Specified Ages

TIME OF BIRTH	DEATHS			
	Number for Age Interval		Percentage for Age Interval	
	20-64	65 and over	20-64	65 and over
1365-1599	191	112	63.0	37.0
1600-1699	311	126	71.2	28.8
1700-1749	313	179	63.6	36.4
1750-1799	710	316	69.2	30.8
1800-1849	1,109	381	77.4	22.6

TABLE 2

Distribution of Observed Deaths for Females of Specified Ages

TIME OF BIRTH	DEATHS			
	Number for Age Interval		Percentage for Age Interval	
	20-64	65 and over	20-64	65 and over
1365-1599	166	147	54.8	45.2
1600-1699	284	192	59.7	40.3
1700-1749	312	215	59.2	40.8
1750-1799	621	418	59.8	40.2
1800-1849	923	484	65.6	34.4

Graduation of the observed ${}_5q_x$ values

The observed ${}_5q_x$ values begin at the age of twenty, which permits graduation by a single curve. The following equation seems to fit the observed data well:

$$\log {}_5q_x = a + bx + cx^2$$

Using the above equation, the observed ${}_5q_x$ values for the age intervals of twenty to seventy-five are graduated by the method of least squares and the graduated ${}_5q_x$ values are determined from the calculated curve. For higher age groups the ${}_5q_x$ values are obtained by extrapolating on the curve. From these graduated ${}_5q_x$ values, the l_x values at each quinquennial age are calculated.

To check the applicability of the above equation in the graduation of the ${}_5q_x$ values, the ${}_5q_x$ values obtained from the United States life table by J. W. Glover for the white males of the Original Registration States for 1910 are graduated using the same curve. The observed and the graduated values are given in Table 3 and plotted in Figure 1, and seem to agree quite well.

TABLE 3

The Observed and Graduated ${}_nq_x$ Values for the White Males of the U. S. Original Registration States for 1910

x	${}_nq_x$ VALUES	
AGE IN YEARS	Observed	Graduated
20	.02615	.02573
25	.02903	.03002
30	.03612	.03579
35	.04521	.04358
40	.05422	.05419
45	.06717	.06883
50	.08428	.08929
55	.11929	.11831
60	.16586	.16012
65	.22845	.22132
70	.31535	.31247
75	.43665	.45057

Computation of $\mathring{e}_x$ values at each quinquennial age

To compute the expectation of life at each quinquennial age, the method sketched below is followed:

Since

$$\mathring{e}_x = \frac{\Sigma L_x}{l_x}$$

(limit of life: x)

and l_x values are already known, $\mathring{e}_x$ can be calculated if the numerator of the right hand expression can be determined. Knowing that

$L_x = \frac{1}{2}(l_x + l_{x+1})$, where L_x denotes the number of persons in the current age interval in the stationary population,

L_x can be obtained by using the Newton formula to the third difference:

$$u = u_0 + x\Delta u_0 + x(x-1)\frac{\Delta^2 u_0}{2} + x(x-1)(x-2)\frac{\Delta^3 u_0}{6}$$

By substituting in the above equation L_x for u, l_x for u_0, and 1/10, 3/10, 5/10, 7/10, and 9/10 for the corresponding values of x, and summing up the L_x values, the following equation is obtained:

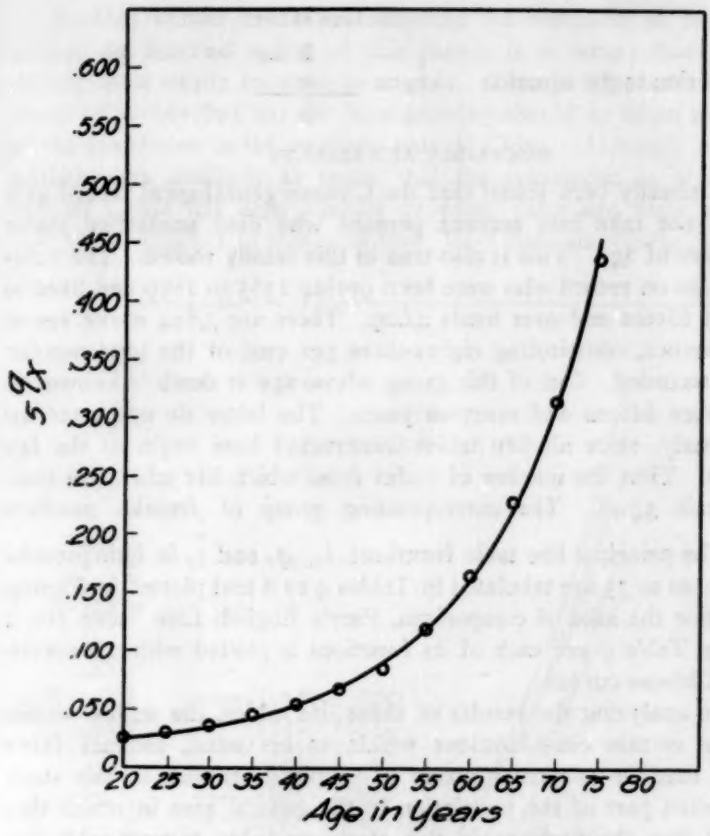

FIG. 1. THE OBSERVED AND GRADUATED $_5q_x$ VALUES.

The small circles are the observed values obtained from Glover's life table for the white males of the U. S. Original Registration States for 1910 and the smoothed line is the fitted curve using the equation

$$\log {}_5q_x = a + bx + cx^2$$

$$\sum_{x}^{x+4} L_x = 5l_x + 2.5\Delta l_x - .425\Delta^2 l_x + .2125\Delta^3 l_x$$

Hence,

$\sum_{x}^{\text{limit of life}} L_x$ can be determined by summing up $\sum_{x}^{x+4} L_x$, thus

giving solution to the equation $\overset{\circ}{e}_x = \frac{\sum_{x}^{\text{limit of life}} L_x}{l_x}$

DISCUSSION AND RESULTS

It has already been stated that the Chinese genealogical record as a rule does not take into account persons who died unmarried under fifteen years of age. This is also true in this family record. The number of males on record who were born during 1365 to 1849 and lived to the age of fifteen and over totals 4,629. There are 3,844 whose age at death is known, constituting eighty-three per cent of the total number of males recorded. Out of this group whose age at death is known, 96 died between fifteen and nineteen years. The latter do not enter the present study, since all life tables constructed here begin at the age of twenty. Thus the number of males from which life tables are computed totals 3,748. The corresponding group of females numbers 3,752. The principal life table functions, l_x, ${}_5q_x$ and $\overset{\circ}{e}_x$ in quinquennial ages from 20 to 75 are tabulated in Tables 4 to 8 and plotted in Figures 2 to 7. For the sake of comparison, Farr's English Life Table No. 1 is given in Table 9 and each of its functions is plotted with the corresponding Chinese curves.

Before analyzing the results of these life tables, the author wishes to present certain considerations which, to his mind, indicate fairly definitely that the particular group of people dealt with in this study are a random part of the population in the general area in which they lived and that the findings in this study probably picture with fair accuracy the mortality conditions in this part of China.

In the first place, what Bell has said about the descendants of the Hyde family in his study of the Hyde genealogy must hold perhaps with more grounds in regard to the people of this Chinese family. The present genealogy covers a much longer period of time and the majority of its males are descendants between the eleventh and twentieth generations. Their blood relationship to their first ancestor is of low order, as is indicated by the fact that he constitutes 1/1024 to 1/65536 of their total ancestry. The females dealt with here can hardly be considered to bear even such blood relationship, as they are all housewives and belong to this family only by virtue of their marriage. Consequently, they must be taken as a sample of the female population in the general locality in which they lived.

Another reason is that on analyzing the condition of reproduction among the married males of this family it is found that they have undergone a steady increase in number. This is important because no group of people that has not been growing should be taken as a sample of the population in the southern part of China. Although no accurate statistics are available to prove that the population in the southern part of China has been steadily growing, there are facts to indicate that the population in this area must have been increasing. First, the

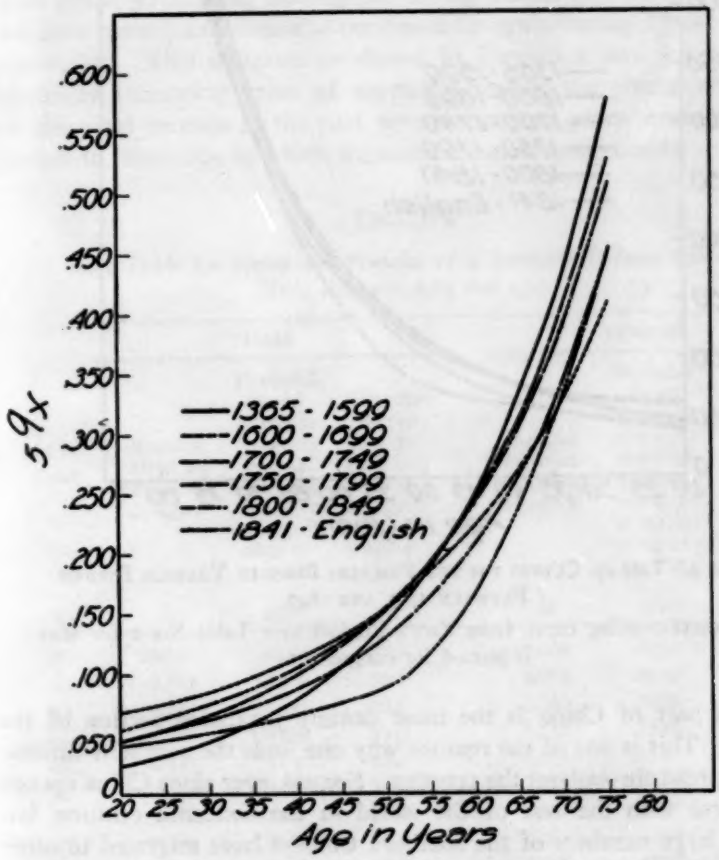

FIG. 2. THE $_5q_x$ CURVES FOR THE MALES BORN IN VARIOUS PERIODS BETWEEN 1365 AND 1849.

The corresponding curve from Farr's English Life Table No. 1 for 1841 is plotted for comparison.

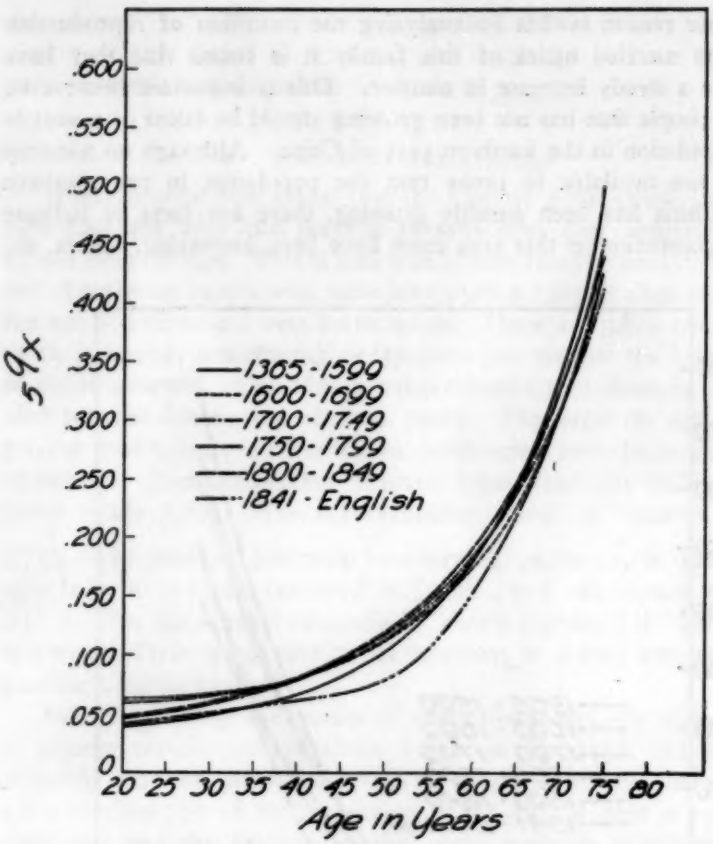

FIG. 3. THE $_5q_x$ CURVES FOR THE FEMALES BORN IN VARIOUS PERIODS BETWEEN 1365 AND 1849.

The corresponding curve from Farr's English Life Table No. 1 for 1841 is plotted for comparison.

southern part of China is the most densely populated section of the country. This is one of the reasons why one finds the southern Chinese widely spread throughout the country. Second, ever since China opened intercourse with the rest of the world in the sixteenth century, immensely large numbers of the southern Chinese have migrated to other parts of the world. The Chinese naturalized in the American countries are practically all from the southern section of China. The same is true of the Chinese migrants in the southern islands in the Pacific Ocean and their numbers are much larger.

In discussing the results, it seems desirable to point out that in view of the limited data the author wishes to emphasize the trend of these life table functions rather than their absolute values. From this viewpoint, the following subjects are considered:

General trend of life table functions

The life tables for both sexes show much higher rates of mortality for the young adult and middle aged group in the period of 1600-1849 than 1365-1599. They are highest during 1600-1699, become lower in the next period, and then rise continuously again during 1750-1799 and 1800-1849. This indicates as shown in Figures 2 and 3 a trend of gradually increasing rates of mortality among the young adult and middle aged persons in the past several hundred years, except for the period of 1600-1699 in which the rates are most unfavorable.

TABLE 4

Life Table for Males and Females of a Southern Chinese Family Born Between 1365 and 1599

AGE IN YEARS	MALE			FEMALE		
	Number alive at beginning of age x	Probability of dying in five-year interval for each one alive at beginning of age x	Expectation of life for each one alive at beginning of age x	Number alive at beginning of age x	Probability of dying in five-year interval for each one alive at beginning of age x	Expectation of life for each one alive at beginning of age x
x	l_x	$_5q_x$	$\overset{\circ}{e}_x$	l_x	$_5q_x$	$\overset{\circ}{e}_x$
20	10,000	.0240	37.7	10,000	.0391	39.7
25	9,760	.0325	33.6	9,609	.0427	36.2
30	9,443	.0440	29.6	9,199	.0480	32.7
35	9,028	.0593	25.9	8,757	.0556	29.2
40	8,493	.0797	22.3	8,270	.0661	25.8
45	7,816	.1068	19.0	7,723	.0810	22.4
50	6,981	.1427	16.0	7,097	.1021	19.1
55	5,985	.1904	13.3	6,372	.1324	16.0
60	4,845	.2521	10.8	5,528	.1767	13.1
65	3,624	.3334	8.6	4,551	.2426	10.3
70	2,416	.4387	6.6	3,447	.3348	7.8
75	1,356	.5779	4.9	2,293	.4979	5.5

TABLE 5

Life Table for Males and Females of a Southern Chinese Family Born Between 1600 and 1699

AGE IN YEARS x	MALE			FEMALE		
	l_x	$_nq_x$	$\overset{\circ}{e}_x$	l_x	$_nq_x$	$\overset{\circ}{e}_x$
20	10,000	.0678	32.9	10,000	.0629	36.9
25	9,322	.0760	30.1	9,371	.0638	34.2
30	8,614	.0860	27.4	8,773	.0667	31.4
35	7,873	.0983	24.8	8,188	.0721	28.5
40	7,099	.1135	22.2	7,598	.0804	25.5
45	6,293	.1323	19.7	6,987	.0926	22.5
50	5,460	.1558	17.3	6,340	.1102	19.5
55	4,609	.1854	15.1	5,641	.1354	16.6
60	3,754	.2227	12.9	4,877	.1718	13.9
65	2,918	.2701	10.9	4,039	.2250	11.2
70	2,130	.3310	9.0	3,130	.3045	8.7
75	1,425	.4096	7.3	2,177	.4254	6.4

TABLE 6

Life Table for Males and Females of a Southern Chinese Family Born Between 1700 and 1749

AGE IN YEARS x	MALE			FEMALE		
	l_x	$_nq_x$	$\overset{\circ}{e}_x$	l_x	$_nq_x$	$\overset{\circ}{e}_x$
20	10,000	.0454	36.6	10,000	.0465	38.0
25	9,546	.0526	33.3	9,535	.0518	34.8
30	9,044	.0618	30.0	9,041	.0587	31.5
35	8,485	.0734	26.8	8,510	.0677	28.3
40	7,862	.0882	23.7	7,934	.0796	25.2
45	7,169	.1073	20.7	7,302	.0953	22.2
50	6,400	.1322	17.9	6,606	.1161	19.3
55	5,554	.1648	15.3	5,839	.1442	16.4
60	4,639	.2080	12.8	4,997	.1823	13.8
65	3,674	.2657	10.5	4,086	.2348	11.3
70	2,698	.3436	8.2	3,127	.3080	9.0
75	1,771	.4497	6.4	2,164	.4115	6.9

TABLE 7

Life Table for Males and Females of a Southern Chinese Family Born Between 1750 and 1799

AGE IN YEARS x	MALE l_x	MALE $_nq_x$	MALE $\mathring{e}_x$	FEMALE l_x	FEMALE $_nq_x$	FEMALE $\mathring{e}_x$
20	10,000	.0449	34.8	10,000	.0476	37.6
25	9,551	.0542	31.3	9,524	.0528	34.3
30	9,033	.0659	27.9	9,021	.0596	31.1
35	8,438	.0808	24.7	8,483	.0688	27.9
40	7,756	.0996	21.7	7,899	.0809	24.8
45	6,984	.1238	18.8	7,260	.0970	21.7
50	6,119	.1548	16.1	6,556	.1187	18.8
55	5,172	.1951	13.6	5,778	.1483	16.0
60	4,163	.2476	11.2	4,921	.1888	13.3
65	3,132	.3163	9.1	3,992	.2453	10.8
70	2,141	.4071	7.2	3,013	.3252	8.5
75	1,269	.5276	5.4	2,033	.4396	6.4

TABLE 8

Life Table for Males and Females of a Southern Chinese Family Born Between 1800 and 1849

AGE IN YEARS x	MALE l_x	MALE $_nq_x$	MALE $\mathring{e}_x$	FEMALE l_x	FEMALE $_nq_x$	FEMALE $\mathring{e}_x$
20	10,000	.0551	33.7	10,000	.0600	36.8
25	9,449	.0638	30.5	9,400	.0617	34.0
30	8,846	.0748	27.4	8,820	.0654	31.1
35	8,184	.0886	24.4	8,243	.0716	28.0
40	7,459	.1061	21.5	7,653	.0809	25.0
45	6,668	.1285	18.8	7,034	.0942	22.0
50	5,811	.1572	16.2	6,371	.1133	19.0
55	4,898	.1945	13.7	5,649	.1405	16.1
60	3,945	.2433	11.5	4,855	.1797	13.3
65	2,985	.3076	9.3	3,983	.2372	10.7
70	2,067	.3931	7.4	3,038	.3229	8.3
75	1,254	.5078	5.6	2,057	.4537	6.0

TABLE 9

Farr's English Life Table No. 1 for 1841

AGE IN YEARS	MALE			FEMALE		
x	l_x	$_1q_x$	$\overset{\circ}{e}_x$	l_x	$_1q_x$	$\overset{\circ}{e}_x$
20	100,000	.04099	39.88	100,000	.04273	40.80
25	95,901	.04647	36.48	95,727	.04716	37.51
30	91,444	.05300	33.13	91,213	.05208	34.24
35	86,597	.05969	29.84	86,463	.05745	30.98
40	81,428	.06756	26.57	81,496	.06335	27.72
45	75,927	.07641	23.31	76,333	.06987	24.42
50	70,125	.08624	20.03	71,000	.07710	21.07
55	64,077	.11952	16.68	65,526	.10540	17.62
60	56,419	.17110	13.59	58,620	.15248	14.39
65	46,766	.24157	10.87	49,682	.21864	11.51
70	35,469	.33457	8.52	38,820	.30823	9.01
75	23,602	.45145	6.55	26,855	.42373	6.91

The political history during the period of 1600-1699 throws light on the extremely high rates of mortality in this century. There was at this time a nation-wide political upheaval in China. At the beginning of the seventeenth century, people rose in the south in revolt against the existing government. Later as the revolutionary movement was gaining strength, the southern revolutionists soon found themselves obliged to face a stronger foe, the rising Manchus who had already conquered north China, established the Manchu government in Peking in 1644 and had set forth to conquer the rest of the country. This struggle lasted for more than half a century, ending in the victory of the Manchus. This undoubtedly had a disastrous effect upon the people of the south, especially the young adult and middle aged persons who were needed either directly or indirectly in carrying out the warfare.

In the succeeding periods, the rates of mortality for the same age group are lower but on the other hand show a definite tendency to rise continuously. In these periods, there are no unusual circumstances operating as during 1600-1699. It is difficult, on the basis of present knowledge, to state just what are the causes of this upward trend of mortality rates among the young adult and middle aged persons in the eighteenth and nineteenth centuries. But this unusual fact is worthy of consideration. For any population, such a trend of increasing rates of mortality in the most energetic and productive period of life is certainly a serious matter, economically and biologically. Although the

present finding is based upon data for people of a particular locality, the fact that it represents one of the richest sections of China indicates the possibility of lower death rates than in most other parts of the country.

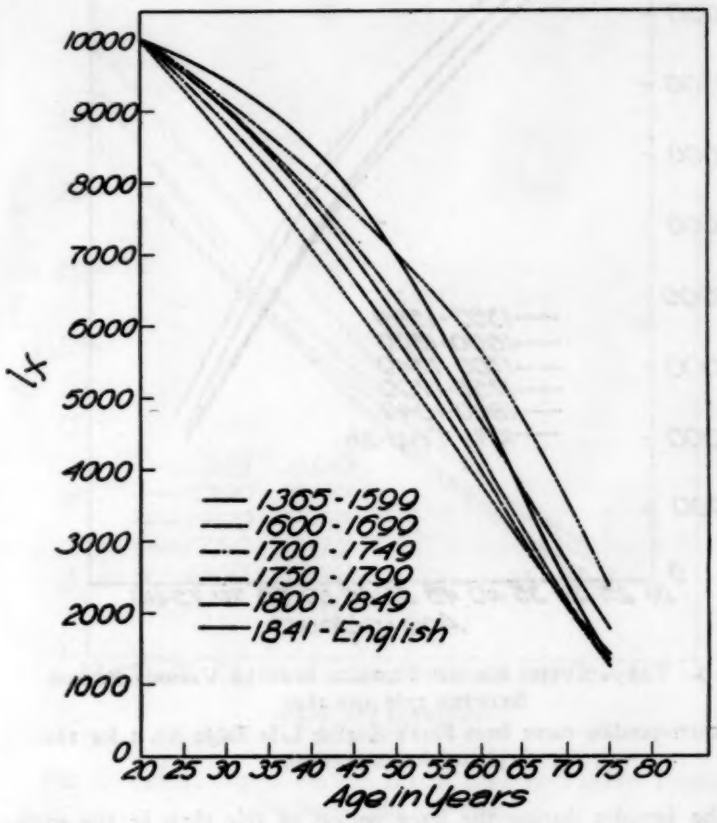

FIG. 4. THE l_x CURVES FOR THE MALES BORN IN VARIOUS PERIODS BETWEEN 1365 AND 1849.

The corresponding curve from Farr's English Life Table No. 1 for 1841 is plotted for comparison.

The survivorship and the expectation of life as shown in Figures 4-7 exhibit a similar trend to the mortality rates for various periods. However, the differences are less marked. This is due to the fact that the mortality rates for higher age groups are more favorable especially

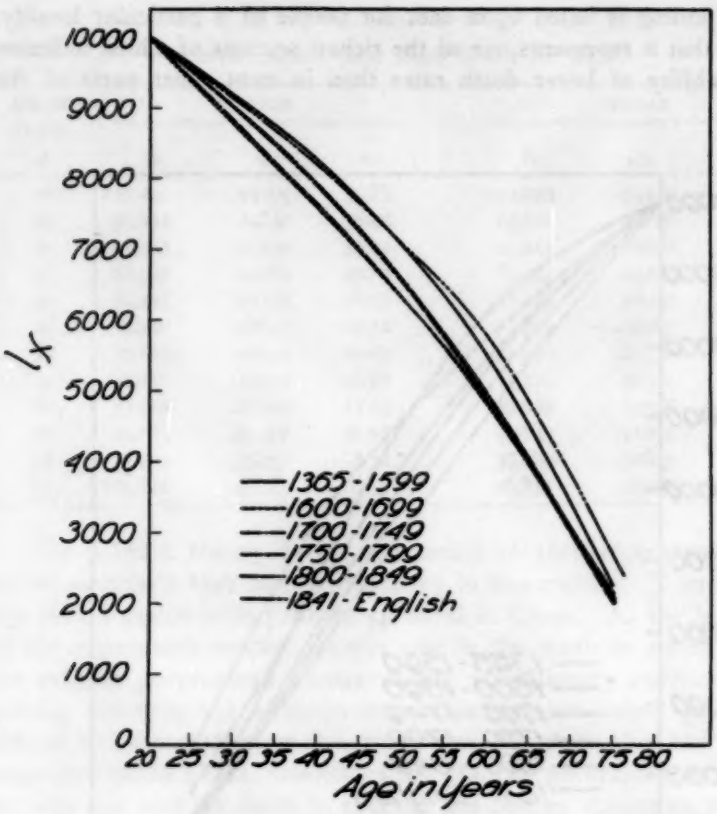

FIG. 5. THE l_x CURVES FOR THE FEMALES BORN IN VARIOUS PERIODS BETWEEN 1365 AND 1849.
The corresponding curve from Farr's English Life Table No. 1 for 1841 is plotted for comparison.

among the females during the later period of life than in the earlier years.

Comparison between males and females

The female mortality rates during various periods are higher than those of the males at the younger ages but lower at the older ages. The only exception occurs during 1600-1699 in which the reverse is true, and this is expected because wartime as a rule is more destructive to the male life. The differences between the two sexes in their age mortality

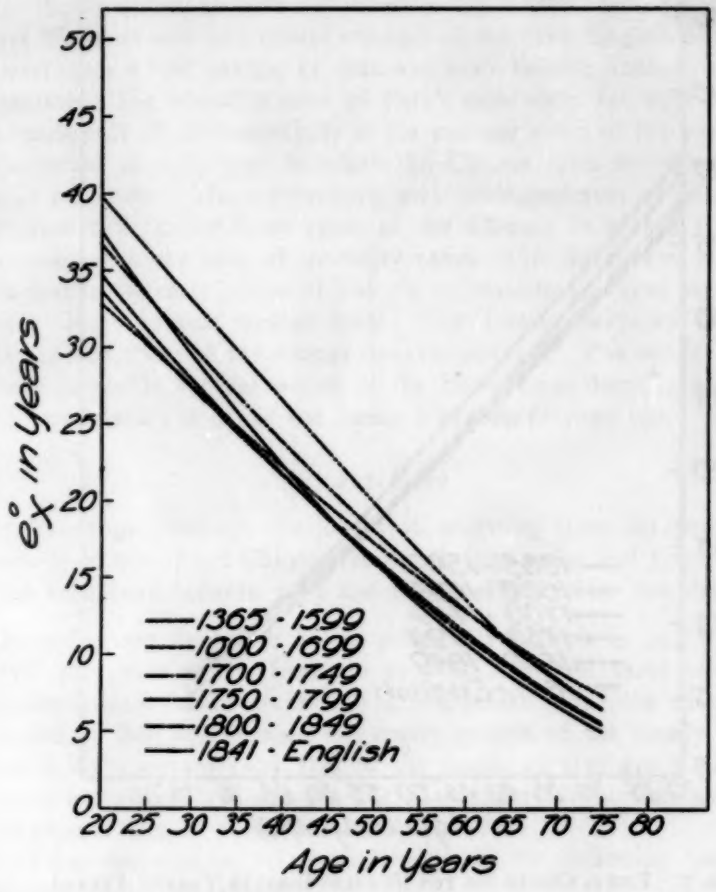

FIG. 6. THE $\mathring{e}_x$ CURVES FOR THE MALES BORN IN VARIOUS PERIODS BETWEEN 1365 AND 1849.

The corresponding curve from Farr's English Life Table No. 1 for 1841 is plotted for comparison.

rates are not peculiar to these life tables, and the same phenomenon is shown in early English life tables except that in the latter the differences are not so striking.

The survivorship of the female life at older ages is far more favorable than that of the male throughout the entire period of 1365-1849. This is explained by the much lower female mortality rates than those of

FIG. 7. THE $\mathring{e}_x$ CURVES FOR THE FEMALES BORN IN VARIOUS PERIODS BETWEEN 1365 AND 1849.

The corresponding curve from Farr's English Life Table No. 1 for 1841 is plotted for comparison.

the male in the later period of life, which outweigh the more favorable male rates at younger ages. For the same reason, the expectation of life is consistently higher in the female than in the male at every age of life and in every period of time.

Comparison with an early English life table

To obtain an idea as to how these Chinese life tables compare with some of the early English life tables, Farr's English life table No. 1

for 1841 is chosen because it is the earliest official life table for England and Wales as well as a typical example of the early English life tables, based upon a vast amount of data and more reliable methods of computation. The mortality rates of Farr's table show for both sexes an advantage at all ages especially at the younger years of life except for the period of 1365-1599 in which the Chinese rates for the younger ages are lower. The survivorship and the expectation of life of the English table differ from those of the Chinese in almost the same manner as in the case of mortality rates. The differences are more marked in the early period of life. It is interesting to note that Farr's table shows several similar facts. The female mortality rates are higher than those of the male at the younger ages. The survivorship is more favorable for the female at the higher ages than for the male. The expectation of life of the female is greater at every age.

SUMMARY

Based upon the age distribution of mortality from the genealogical records of a southern Chinese family of 3,748 males and 3,752 females who were born between 1365 and 1849, ten life tables have been constructed giving the values of the principal functions, l_x, ${}_5q_x$, and $\mathring{e}_x$ at each quinquennial age from 20 to 75. Although these tables are obtained from family records, it is not unlikely, for the reasons advanced by Bell and because the steady growth of the family population is in agreement with that of the people of southern China, that they picture with fair accuracy the condition of the Chinese people in the general area in which this family has lived.

From the analysis of these life tables, the following facts may deserve special emphasis:

1. The outstanding finding in this investigation is the trend of gradually rising death rates among the young adult and middle aged life of both sexes over the period 1365-1849, except for the century 1600-1699 in which the rates are aggravated by warfare. The same trend holds in the survivorship as well as the expectation of life. This indicates that in the past several centuries the most energetic and productive persons have been dying faster and faster and their expectation of life has become shorter and shorter.
2. The female mortality rates are higher than the male at younger ages but lower at older ages, except in the period of revolution and warfare. The survivorship at older ages is considerably more favorable for the female life. For the same reason, the female

expectation of life is consistently higher at every age and in every period of time.

3. In comparing these Chinese life tables with Farr's English life table No. 1, the latter shows for both sexes lower mortality rates, more favorable survivorship and greater expectation of life practically at all ages and especially at the younger ages, except for the period of 1365-1599 in which the Chinese mortality rates are lower. It is worth noting that the English and Chinese life tables exhibit several points of similarity. Both tables indicate among the females more favorable survivorship at the higher ages and greater expectation of life at every age in spite of the higher female mortality rates at the younger ages.

APPENDIX

OBSERVED DATA FROM WHICH LIFE TABLES ARE CONSTRUCTED

TABLE 10

Observed d_x, l_x and $_nq_x$ Values for Both Sexes Born Between 1365 and 1599

AGE IN YEARS x	MALE			FEMALE		
	d_x	l_x	$_nq_x$	d_x	l_x	$_nq_x$
20	9	1,000	.0300	10	1,000	.0330
25	10	870	.0340	12	967	.0414
30	11	937	.0384	17	927	.0604
35	13	901	.0477	19	871	.0712
40	22	858	.0851	15	809	.0618
45	23	785	.0955	14	759	.0606
50	36	710	.1676	27	713	.1248
55	38	591	.2132	21	624	.1122
60	29	465	.2043	31	554	.1841
65	46	370	.4108	31	452	.2257
70	27	218	.4083	36	350	.3400
75	22	129	.5659	38	231	.5411
80	13	56	....	20	106	....
85	4	13	....	8	40	....
90	..	..	....	2	13	....
95	..	..	....	1	7	....
100	..	..	....	1	3	....
Total	303	..	....	303	..	....

TABLE 11

Observed d_x, l_x and $_nq_x$ Values for Both Sexes Born Between 1600 and 1699

AGE IN YEARS	MALE			FEMALE		
x	d_x	l_x	$_nq_x$	d_x	l_x	$_nq_x$
20	21	1,000	.0480	34	1,000	.0710
25	38	952	.0914	27	929	.0614
30	46	865	.1214	19	872	.0459
35	37	760	.1118	31	832	.0781
40	31	675	.1052	40	767	.1095
45	29	604	.1093	32	683	.0981
50	33	538	.1413	31	616	.1071
55	38	462	.1883	32	550	.1218
60	38	375	.2320	38	483	.1656
65	31	288	.2465	41	403	.2134
70	31	217	.3272	48	317	.3186
75	29	146	.4521	45	216	.4352
80	22	80	....	35	122	....
85	8	30	....	15	48	....
90	4	11	....	4	17	....
95	1	2	....	4	8	....
Total	437	...	....	476	...	....

TABLE 12

Observed d_x, l_x and $_nq_x$ Values for Both Sexes Born Between 1700 and 1749

AGE IN YEARS	MALE			FEMALE		
x	d_x	l_x	$_nq_x$	d_x	l_x	$_nq_x$
20	23	1,000	.0470	23	1,000	.0440
25	28	953	.0598	20	956	.0397
30	22	896	.0491	36	918	.0741
35	30	852	.0716	34	850	.0753
40	36	791	.0936	45	786	.1094
45	38	717	.1074	28	700	.0757
50	45	640	.1422	45	647	.1314
55	36	549	.1330	44	562	.1495
60	55	476	.2353	37	478	.1464
65	60	364	.3352	35	408	.1618
70	35	242	.2934	62	342	.3450
75	37	171	.4386	60	224	.5089
80	30	96	....	33	110	....
85	12	35	....	16	47	....
90	5	10	....	6	17	....
95	..	..	....	2	6	....
100	..	..	....	1	2	....
Total	492	..	....	527	..	....

TABLE 13

Observed d_x, l_x and $_nq_x$ Values for Both Sexes Born Between 1750 and 1799

AGE IN YEARS	MALE			FEMALE		
x	d_x	l_x	$_nq_x$	d_x	l_x	$_nq_x$
20	48	1,000	.0470	43	1,000	.0410
25	61	953	.0619	58	959	.0584
30	54	894	.0593	58	903	.0620
35	50	841	.0583	77	847	.0874
40	92	792	.1124	51	773	.0634
45	104	703	.1451	86	724	.1146
50	88	601	.1414	71	641	.1061
55	110	516	.2093	79	573	.1326
60	103	408	.2451	98	497	.1911
65	108	308	.3409	99	402	.2363
70	84	203	.4039	117	307	.3681
75	61	121	.4959	87	194	.4278
80	46	61	....	65	111	....
85	10	17	....	42	48	....
90	6	7	....	7	8	....
95	1	1	....	1	1	....
Total	1,026	..	....	1,039	..	....

TABLE 14

Observed d_x, l_x and $_nq_x$ Values for Both Sexes Born Between 1800 and 1849

AGE IN YEARS	MALE			FEMALE		
x	d_x	l_x	$_nq_x$	d_x	l_x	$_nq_x$
20	78	1,000	.0520	82	1,000	.0580
25	111	948	.0791	90	942	.0679
30	110	873	.0848	107	878	.0866
35	112	799	.0939	95	802	.0848
40	134	724	.1243	99	734	.0954
45	130	634	.1372	98	664	.1054
50	153	547	.1883	98	594	.1162
55	130	444	.1959	108	525	.1467
60	151	357	.2829	146	448	.2321
65	134	256	.3516	131	344	.2703
70	128	166	.5181	121	251	.3426
75	65	80	.5750	113	165	.4848
80	39	36	....	73	85	....
85	12	10	....	34	33	....
90	2	2	....	10	9	....
95	0	1	....	1	2	....
100	1	1	....	1	1	....
Total	1,490	..	....	1,407	..	....

TABLE 15

Observed d_x, l_x and $_nq_x$ Values Adjusted for Incompleteness at Age of 65 Years and Over for Both Sexes Born Between 1800 and 1849

AGE IN YEARS	MALE			FEMALE		
x	d_x	l_x	$_nq_x$	d_x	l_x	$_nq_x$
20	78	1,000	.0490	82	1,000	.0530
25	111	951	.0726	90	947	.0634
30	110	882	.0782	107	887	.0767
35	112	813	.0849	95	819	.0745
40	134	744	.1129	99	758	.0844
45	130	660	.1127	98	694	.0922
50	153	579	.1658	98	630	.1000
55	130	483	.1677	108	567	.1235
60	151	402	.2338	146	497	.1911
65	↑	308	.3409	↑	402	.2363
70	|	...	.4039	|	...	.3681
75	|	...	.4959	|	...	.4278
80						
85	494	...	....	621	...	....
90	|	...	....	|	...	....
95	|	...	....	|	...	....
100	↓	...	....	↓	...	....
Total	1,603	...	....	1,544	...	....

THE CORRELATION BETWEEN GROWTH RATES OF POPULATION AND VITAL FACTORS BY GEOGRAPHICAL DISTRICTS*

BY HARUO MIZUSHIMA

INTRODUCTION

Population growth is one of the most important social problems of the present day. Being intimately correlated with industrial development, it has been earnestly discussed from the economic and sociological points of view. And since the problem arises as a consequence of aggregative life of human beings, a kind of living organism, it has been investigated as a biological phenomenon.

Population growth is influenced by such extremely complicated economical and socio-biological conditions that it is obviously unreasonable to attempt to grasp the entirety of the problem from a single standpoint. It must be approached and attacked from every angle possible.

The purpose of this paper is not of course to treat thoroughly such a vast field as population growth, but to touch the problem at the point where population growth is correlated with socio-biological factors which are submissible to statistical treatment.

METHOD AND MATERIAL

To investigate the relationship between growth-rate of population and vital factors which may influence it, there are two principal methods, the one historical and the other geographical. If the development of population of any community is followed historically for a sufficiently long period of time, it may be revealed that the growth-rate of population has not been uniform throughout the whole period, but has fluctuated considerably, being rapid at some periods and slow or even negative at others. The comparative investigation of the underlying elements

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The writer desires to acknowledge his indebtedness to Doctor Lowell J. Reed for suggestions and criticism received during the prosecution of this study.

which have determined the rapidity of the population growth is the historical method. This method requires of course ample statistical records for a long period of time, which are usually unavailable. The geographical method consists in comparing the growth-rate of population in various districts of a community divided geographically, and investigating the vital factors which are responsible for causing the difference of growth-rates of population.

The procedure of investigation adopted in this paper is the geographical method. The growth-rate of each state in the United States and of each prefecture in Japan was first determined, and then the correlations between these rates and various vital factors were examined statistically.

Although the United States and Japan are markedly different, the social conditions are alike in many respects. The population growth in both countries also presents many similar as well as different aspects. Therefore it seems interesting in discussing the present problem to compare the situations in the two countries.

First of all, the rate of population growth of each state or prefecture must be determined. In the United States the census reports since 1790 supply the necessary data concerning the population of each state.

As Pearl and Reed[1,2] demonstrated, population growth can be satisfactorily expressed by the logistic equation. But if we confine the argument to periods of time so short as four or five decades (since the Civil War), the population growth of each state can be expressed without great discrepancy by the simple exponential equation in such a general form as

$$y = e^{a+bx}$$

where y is the population at any year x, x is the time in years counted from an arbitrary origin, a is a constant to be determined according to the population at the arbitrary origin, and b is a constant which indicates the rate of population increase, being large or small according as the population increase is rapid or slow.

This exponential equation was fitted to the population growth of each state by the method of least squares, using logarithmic residuals. In a few states, Oklahoma, Nevada and Nebraska, the population increase is too irregular to be fitted by this equation; consequently these

[1] Pearl, R. The curve of population growth. Studies in Human Biology, pp. 558-583.

[2] Pearl, R. The growth of human population. *Id.*, pp. 584-637.

states were excluded. North and South Dakota were treated as a single unit, the population of the two states being added together.

The primarily important constant in the above equation is b, which shows the growth-rate of population. The value of the b constant computed for each state is shown in Table 1.

In Japan, the first national census was carried out in 1920 and the second in 1925. Before that time the population had been estimated by means of residence registration, which showed a tendency to be overestimated and consequently was not as accurate as the census count. As it seemed to be rather unreasonable to fit exponential equations to these different kinds of reports, the two latest census reports only were used and the rate of population growth of each prefecture was computed on the assumption of geometric increase. The growth-rate thus computed is shown in Table 2. These constants may be taken as indicating the average rate of growth of the population in the prefectures over the period 1920 to 1925.

Looking at these tables, it will be perceived that the values of the b constant vary considerably. In the United States, b's are high in the mountain and Pacific regions where settlement is recent, the density of population sparse and wide areas of land uncultivated. On the contrary, b's are very small in some states in New England such as Maine, New Hampshire and Vermont.

In Japan six prefectures, wherein large cities such as Tokyo, Osaka, Kyoto, Nagoya, Kobe, and Yokohama are situated, have high values of b. As these prefectures are urban and have many peculiar characteristics differing from other parts of Japan, it is frequently necessary to exclude them from the general discussion. Of the rural prefectures the northeastern districts have relatively high values of b. Generally speaking, b's are small in prefectures facing the Japan Sea, and large in those along the Pacific Coast. A somewhat exceptional case is Okinawa-kin (Loochoo Island), which has its own characteristic social features and whose population is decreasing. Hence it is not included in the discussion of the other districts of Japan.

TRUE RATE OF NATURAL INCREASE OF POPULATION

Dublin and Lotka [3] introduced the conception of true rate of natural increase of population in 1925. Here I wish to sketch the outline of their method and to report some of my results obtained by applying it.

[3] Dublin, L. I., and Lotka, A. J. On the true rate of natural increase. *Jour. Amer. Stat. Assoc.*, vol. XX, Sept., 1925.

Usually the excess of births over deaths (per 1000 persons) is considered as the rate of natural increase of population. This is right if there has been no migration and the birth-rate and death-rate have been kept unchanged for the past several decades at least. Under such conditions the age constitution of the population is stationary, and the excess of birth-rate over death-rate shows the true rate of natural increase.

The United States had in the recent past free immigration and a higher birth-rate than it has today. These facts caused a relative swelling in the numbers of middle-aged persons. But owing to the restriction of immigration on the one hand, and the impossibility of continuance of decline in the birth-rate on the other hand, the present proportion of the age distribution of the population cannot remain constant.

The true rate of natural increase (ρ)* is the excess of birth-rate over death-rate under the condition that the age constitution of the population is stationary, which would occur if the age-specific birth- and death-rates became constant and migration ceased.

The United States is now in a very favorable condition from the viewpoint of age constitution, but this condition cannot be permanent. Hence, in the United States the true rate of natural increase is quite different from the actual.

This can be computed by solving the equation

$$1 = \int_0^\infty e^{-\rho a}\, p(a)\, m(a)\, da$$

where a is any arbitrary age, $p(a)$ is the probability at birth that a random individual will reach age a, and $m(a)$ is the maternity frequency per annum of females of age a. Applying this method and utilizing the data of 1920, ρ for each birth registration state was computed (Table 1).

It is to be regretted that the data of Japan are not available for carrying out the same procedure as in the United States, and in this respect it is impossible at present to compare the two countries.

Now, what relationship does this true rate of natural increase have to the actual rate of population growth and to other vital factors? In the following discussion this question is interwoven with the relationships between the actual growth-rate and vital factors.

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* r was originally used by Dublin and Lotka as the symbol of true rate of natural increase. But here ρ is used instead, in order to avoid confusion since r is commonly used as the symbol of the coefficient of correlation.

First of all, the correlation between the actual growth-rate (b) and ρ is as follows:

$r_{b\rho} = +.0363 \pm .1561$

This coefficient is entirely insignificant and shows that, so far as the data of 1920 are concerned, the true rate of natural increase was uncorrelated with the actual population growth in these birth registration states.

BIRTH-RATE

Since births contribute to the increase of population, birth-rate (B) may be expected to have positive correlation with the growth-rate (b) of population. In the United States the correlation coefficient between b and the annual average of birth-rates (B) over the period 1915-1920 in twenty-one birth registration states is

$r_{bB} = +.5233 \pm .1124$

If the data in the year 1920 only are used,

$r_{bB} = +.4605 \pm .1219$

Before birth registration was introduced, the reports on birth-rates are available in the census year only.

$r_{bB'} = +.5488 \pm .0745$ 1900

$r_{bB'} = +.5248 \pm .0764$ 1890

where B' is the birth-rate per 1000 females.

These coefficients are all positive and significant. From this result we know that the birth-rates are, as expected, relatively higher in states growing more rapidly in population.

In Japan the situation is almost like that of the United States, except for six urban prefectures where the population growth is extraordinarily rapid and the birth-rates are very low. The correlation coefficients between b and the birth-rates are as follows:

$r_{bB} = +.5200 \pm .0788$ . . . . 1920-1922, 40 rural prefectures

$r_{bB} = +.4538 \pm .0863$ . . . . 1923-1925, 40 rural prefectures

This result also shows that the birth-rate is positively correlated with population growth.

Further light on this correlation may be had by considering the birth-rates (β) per 1000 married women of age 15-44 years instead of the crude birth-rates. In this case the coefficients of correlation turned out to be

In the United States . . . . . $r_{b\beta} = +.2534 \pm .1317$

where β is the annual average for 1920-1921.

In Japan . . . . . $r_{b\beta} = +.3878 \pm .0924$

where β is the annual average for 1924-1926.

These coefficients are lower than in the previous case, in the United States almost insignificant, in Japan still significant. These barely significant correlations are due, as indicated by Pearl [4] to the complexity of various social conditions such as education, wealth, density of population, occupation, and in the United States, the percentage of foreign-born population, which is relatively more numerous in states growing rapidly and has more vigorous reproductive power than native population.

On the other hand, the birth-rates per 1000 married females of age 15-44 years have a very high correlation with the true rate of natural increase (ρ) since

$$r_{\rho\beta} = +.9395 \pm .1561$$

The reason is obvious when we consider the derivation of ρ. The low correlations between (b), the growth-rate of the population, and (β) the birth-rate per 1000 married females, is in harmony with the low correlation previously determined between b and ρ.

DEATH-RATE

Since death means diminution in the number of living individuals, it may be expected that the death-rate is negatively correlated with the growth-rate of population. The coefficients of correlation between the crude death-rate (D) and population growth (b) are as follows:

In the United States:

$r_{bD} = -.5938 \pm .0760$ for 33 death registration states, 1911-1920

$r_{bD} = -.3750 \pm .1159$ for 27 death registration states, excluding the Mountain and Pacific regions, 1911-1920

$r_{bD} = -.5492 \pm .1014$ for 19 death registration states, 1906-1910

$r_{bD} = -.2766 \pm .1157$ for 30 states, 1880

$r_{bD} = -.3342 \pm .0936$ for 41 states, 1870

In Japan:

$r_{bD} = -.1976 \pm .0966$ for all Japan, 1920-1922

$r_{bD} = -.200 \pm .0955$ for all Japan, 1923-1925

These results show that in the United States the correlation is negative and significant, but in Japan, though negative, practically insignificant.

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[4] Pearl, R. Biology of Population Growth. Williams and Wilkins Press, Baltimore, Md.

This latter fact suggests that in Japan the birth-rate is playing a more important rôle in population growth than the death-rate.

This low correlation between the death-rate and population growth in Japan can be explained by the fact that districts where b's are comparatively high have relatively high birth-rates and high infant mortality rates; consequently the total crude death-rate cannot be low. Therefore, if the infant deaths are excluded from the total deaths, and the crude death-rates of people above one year are used, the correlation coefficient becomes larger, as

$r_{bD} = -.7346 \pm .0463$ . . . . . all Japan.

This result shows obviously that the death-rates of persons above one year are comparatively lower in those prefectures where the population is increasing more rapidly.

Is the smaller size of the death-rate in rapidly growing districts due to the better sanitary conditions or to some other factors? To answer this question, first of all the age constitution of population must be taken into consideration, and the death-rates must be corrected accordingly. Paying attention to this point, the correlation coefficients between the growth-rate (b) and the adjusted death-rate were found to be as follows:

In the United States:

$r_{bR} = -.3129 \pm .1092$

where R is the average adjusted death-rates of 33 birth registration states for 1920-1921. This coefficient is lower than in the case of the crude death-rates, but still slightly significant. But if the Mountain and Pacific regions are eliminated, because the states in these districts have extraordinarily high values of b and have consequently a tendency to exaggerate the result, then the correlation coefficients between b and the crude and adjusted death-rates of twenty-seven states in the middle and eastern parts of the country are as follows:

$r_{bD} = -.3750 \pm .1395$ . . . . . crude death rates

$r_{bR} = -.0204 \pm .1323$ . . . . . adjusted death rates

The former is still slightly significant, but the latter certainly insignificant.

In Japan, if the adjusted death-rates [5] are used

$r_{bR} = -.0186 \pm .1006$ . . . . . all Japan, 1925

This coefficient is beyond doubt insignificant, too.

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[5] Imai, T. On the death-rates of large cities and rural districts in Japan. (Japanese text.) *Chirigaku Hyoron*, Apr., 1929.

These results indicate that the negative correlations between b and the crude death-rates are due mainly to the difference of age constitution of the population and not to the difference of the sanitary conditions.

The correlations between the true rate of natural increase (ρ) and the crude or adjusted death-rate are found to be as follows:

$r_{\rho D} = -.2204 \pm .1561$ . . . . . crude death-rate, 1920

$r_{\rho R} = -.0509 \pm .1561$ . . . . . adjusted death-rate, 1920-1921

These coefficients are practically insignificant. Or in other words, there is almost no relationship between the true rate of natural increase and either of the death-rates. This result shows that the true natural increase of population is mainly dependent on the birth-rate and is very little affected by the death-rate.

INFANT MORTALITY

In the United States infant mortality is negatively correlated with population growth since

$r_{bM} = -.4881 \pm .1246$

where M is the annual average of infant death-rates per 1000 births in nineteen birth registration states for 1919-1920, the Pacific region being eliminated. In these states the birth-rates are positively correlated with population increase

$r_{bB} = +.3649 \pm .1418$

Keeping the birth-rate constant, the coefficient of partial correlation between M and b is

$r_{bM.B} = -.5487 \pm .1145$

which is significant. Judging from this result, we may say that infant mortality is comparatively lower in states growing more rapidly in population.

In Japan the infant mortality has no correlation with population growth simply because those prefectures in which the b's are large have not necessarily lower infant mortality on account of the relatively higher birth-rates. And it can be seen from the following coefficient that the true rate of natural increase (ρ) is uncorrelated with the infant mortality (M)

$r_{\rho M} = +.1652 \pm .1561$

VITAL INDEX AND EXCESS OF BIRTHS OVER DEATHS

Since birth-rate is positively, and death-rate is negatively, correlated with population growth, vital index ($V = \frac{\text{births}}{\text{deaths}} \times 100$) and excess of

births over deaths ($X = \frac{\text{births—deaths}}{\text{population}} \times 1000$) must naturally be expected to have positive correlation with population growth. The results are as follows:

In the United States:

$r_{bV} = +.6139 \pm .0893$

$r_{bX} = +.5982 \pm .0903$

where V and X are the annual average of vital indices and the excess of birth-rates over death-rates respectively for 1919-1920 in nineteen birth registration states.

In Japan the situation is practically the same:

$r_{bV} = +.5399 \pm .0770$

$r_{bX_1} = +.6030 \pm .0687$

$r_{bX_2} = +.6744 \pm .0589$

where V is the annual average of vital indices for 1923-25 in all Japan and X_1 and X_2 are those of excess of birth-rates over death-rates for 1920-1922 and 1923-1925 respectively in forty rural prefectures (Table 2). These coefficients are all significant as was to be expected.

The excess of crude birth-rates over death-rates (X) and the vital index have the following correlation coefficients with the true rate of natural increase (ρ):

$r_{\rho X} = +.9591 \pm .1561$

$r_{\rho V} = +.8856 \pm .1561$

These are extraordinarily high correlations, especially the former one, which is very near to perfect correlation. This result shows that, although the difference between birth-rate and death-rate does not represent the true rate of natural increase, it still does form an index of natural increase that will give the relative rating of different states with great accuracy.

MIGRATION

It is a matter of course that the rapid increase of population in the United States has owed not a little to immigration from foreign countries. And the population of each state has also been affected, more or less, by immigration. The correlation between the growth-rates of population (b) and the percentages of foreign-born population (f) is as follows:

$r_{bf} = +.3171 \pm .0931$ 1920

$r_{bf} = +.3826 \pm .0868$ 1910

This result shows that in states having a large b, there are relatively more foreign-born people. Since immigration contributes to the increase of actual number of population, the result obtained here is naturally to be expected.

Besides this actual increase in number of population, there is a certain biological factor affecting the population growth. Pearl [6] indicated that the vital index of foreign-born whites (B) had always been higher than that of the native whites (A), as the following table shows:

	1915 Vital Index		1916 Vital Index		1917 Vital Index		1918 Vital Index	
	A	*B*	*A*	*B*	*A*	*B*	*A*	*B*
Cities	100.5	267.5	100.5	247.8	117.0	228.3	93.2	166.9
Rural	141.1	215.4	137.7	199.9	177.7	156.5	144.8	118.8
Total	117.8	252.4	116.3	234.1	148.1	205.2	118.8	151.8

Whelpton [7] showed by applying Dublin and Lotka's method that the true rate of natural increase of foreign-born population is much higher than the native, as can be seen from the following table:

	ρ
Native whites	
Industrial states (Conn., Mass., N. Y.)	−6.0
Agricultural states (Ky., N. C., S. C.)	20.2
11 large cities	7.9
Urban districts	−5.6
Rural districts	17.8
Foreign whites	
Industrial states	13.6
Agricultural states	19.3
11 large cities	11.7
Negroes	
7 southern states	13.0
10 northern states	−4.8

From these investigations it must be deduced that the high percentages of foreign-born population are accompanied with high power of reproduction. It might be said that this is an indirect contribution of immigration to the population growth.

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[6] Pearl, R. The vitality of the peoples of America. *Amer. Jour. Hyg.*, vol. I, pp. 592-674, 1921.

[7] Whelpton, P. K. Differentials in true natural increase. *Jour. Amer. Stat. Assoc.*, vol. XXIV, Sept., 1929.

Judging from the above deduction, it might be expected that the true rate of natural increase (ρ) would be positively correlated with the percentages of foreign-born population, but in reality it turns out to be

$$r_{\rho f} = -.6114 \pm .1561$$

This is definitely negative correlation and shows that in places where foreign-born populations are more numerous, the true rate of natural increase tends to be lower. This is apparently paradoxical, but this paradox can for the most part be explained by the fact that the immigrants from the foreign countries have a strong tendency to gather in the urban districts rather than in the rural. And as will be discussed later, the true rates of natural increase are relatively lower in the urban sections than in the rural. These are the principal factors which cause the correlation between f and ρ to be negative; this is confirmed by the fact that the partial correlation between these two items becomes much lower, practically insignificant, if the proportion of rural population is kept constant:

$$r_{\rho f.R} = +.2013 \pm .1561$$

The amount of immigration and emigration between states has considerable influence on the population growth of each state. The correlation coefficients between the percentages of such immigrants (i) and of emigrants (e) with b are as follows:

$r_{bi} = +.7393 \pm .0461$	. . . . .	1920
$r_{bi} = +.7290 \pm .0476$	. . . . .	1910
$r_{be} = -.3802 \pm .0960$	. . . . .	1910-1920
$r_{be} = -.4563 \pm .0805$	. . . . .	1900-1910

All these coefficients are significant, and we can see that states growing more rapidly in population have comparatively more native immigrants from other states and fewer emigrants to other districts.

The population growth of each state depends obviously on the following three principal elements:

1. The natural increase of population born in the state of residence;
2. The immigrants from other states;
3. The immigrants from foreign countries.

We have seen that each of these elements is positively correlated with the actual growth-rate of each state. But the question may be raised as to which one of these elements is playing the most important rôle in influencing the actual population growth of each state. This is a problem of sufficient interest to be considered somewhat in detail;

hence the growth-rate of the following elements of the population was determined:

1. Population born and living in the specified state;
2. Population born in other states and living in the specified state;
3. Population born in foreign countries and living in the specified state.

An exponential equation was fitted by the method of least squares using logarithmic residuals, to the growth of each element of population described above in each state from 1870 or 1880 to 1920 and the values of b constants of $y = e^{a+bx}$ were determined, designated by b_1, b_2 and b_3 respectively (see Table 1). These constants indicate the rates of growth, b_1 being the rate of increase of population born and living in specified state, b_2 being the rate of increase of population born in other states and living in specified state, and b_3 being the rate of increase of population born in foreign countries and living in specified state.

Next, the correlation coefficients between b (growth-rate of total population) and b_1, b_2 and b_3 respectively were computed. The results are as follows:

$$r_{bb_1} = +.9189 \pm .0160$$
$$r_{bb_2} = +.7484 \pm .0452$$
$$r_{bb_3} = +.7140 \pm .0504$$

These coefficients are all highly significant, r_{bb_1} being the highest. This result indicates that the growth-rate of population born in the state of residence is most closely correlated with that of the total population. In order to confirm this conclusion, the partial correlations must be taken into consideration, because these b constants are intimately correlated with one another. The coefficients thus computed are as follows:

$$r_{bb_1.b_2b_3} = +.9150 \pm .0166$$
$$r_{bb_2.b_1b_3} = +.5915 \pm .0661$$
$$r_{bb_3.b_1b_2} = +.4227 \pm .0835$$

From these results we can conclude with confidence that the growth-rate of total population by states is most closely correlated with that of population born in the state of residence and least with that of foreign-born population.

In Japan, the influence of international migration on the total population growth has been practically negligible, and will not be touched upon here. But from the viewpoint of population increase by prefectures, immigrants from and emigrants to other districts must be

considered. The correlation coefficient between the growth-rate (b) and the percentage of such immigrants (i) is as follows:

$r_{bi} = +.6430 \pm .0583$ . . . . . 1920, all Japan

This high coefficient is due mainly to the six urban prefectures, which have high growth-rates and a large number of immigrants. If these six prefectures are eliminated, the coefficient becomes so small as to be practically insignificant:

$r_{bi} = +.1350 \pm .0976$

This result states that in the rural prefectures immigrants from other districts have almost no influence on the growth-rate of population.

On the other hand, the correlation between b and the percentage of emigrants to other districts is as follows:

$r_{be} = -.5647 \pm .0677$ . . . . . 1920, all Japan

$r_{be} = -.5417 \pm .0703$ . . . . . 1920, 40 rural prefectures

In both cases the coefficients are highly significant and show that the actual growth-rate is negatively correlated with emigration to other districts, mostly to large cities. Moreover, we can say that in the rural districts the population is not increased appreciably by immigration from other prefectures, but is damped significantly by emigration to the urban districts.

RURAL POPULATION—AGRICULTURE

In the United States there is no definite correlation between the actual growth-rate of population by states and the percentage of rural population (R). But the latter has a significant positive correlation with the true rate of natural increase (ρ) as can be seen by the following coefficient:

$r_{\rho R} = +.6387 \pm .1500$

where R is the percentage of rural population in nineteen states, 1920. This result shows clearly that in the rural districts the natural increase is higher than in the urban and coincides exactly with Whelpton's result which was cited previously.

Similarly, the percentage of farmers of 10 years of age and over (A) is positively correlated with ρ:

$r_{\rho A} = +.6606 \pm .1561$

This would indicate that the rural population is contributing a relatively large share to the natural increase of population in the United States.

DENSITY OF POPULATION

By many vital statisticians density of population has been claimed to exert an important influence on reproduction and mortality. Farr's law attempted to express the relation between density of population and death-rate. LeBlanc [8] investigated this relation with the census reports of the United States for 1910 but failed to confirm Farr's law. The writer also has not been able to obtain any correlation between the population density and corrected death-rates of forty-six prefectures, and the crude death-rates of ninety-seven cities in Japan in 1925.

On the other hand, certain relationships were found between population density and reproduction in Japan, as well as in the United States. In dealing with density of population, however, it will almost always be found that its frequency distribution is very skew, sparsely populated districts being much more numerous than densely populated ones. This fact renders the interpretation of the coefficient of correlation very difficult.

This sort of difficulty can be avoided if the logarithm of density is used instead of its actual figures (persons per square mile). This simple manipulation will in usual cases make the frequency distribution of density practically normal, and the interpretation of the result easier.

In the United States, the correlation coefficient between the birth-rates (β) per 1000 married females of age 15-44 years for 1920-1921 in birth registration area and the logarithm of population density (l) in 1920 is as follows:

$$r_{l\beta} = -.3817 \pm .1359$$

The coefficient is less than three times its probable error and cannot be claimed to be very significant.

In Japan the same kind of correlation coefficient for forty-six prefectures in 1925 is

$$r_{l\beta} = -.5599 \pm .0675$$

Using the crude birth-rates in ninety-seven cities

$$r_{lB} = -.3192 \pm .0615$$

where B is birth-rate per 1000 population. These coefficients are fairly significant. This result indicates that reproduction is relatively weaker in densely populated districts.

If all states are taken into account, no correlation can be observed between the growth-rates (b) and density of population (l). But if the

[8] Le Blanc, T. J. Density of population and mortality in the United States. *Amer. Jour. Hyg.*, vol. IV, pp. 501-558, 1924.

rural states only (by rural is meant here such a population distribution that more than fifty per cent of all population is living in communities of less than 5,000 persons) are considered, there exists a certain definite relationship between b and l, as the following coefficients show:

$r_{bl} = -.7140 \pm .0623$ . . . . . 1920, 29 states
$r_{bl} = -.7496 \pm .0522$ . . . . . 1910, 33 states

This result states that the sparser the population, the more rapid is the growth of population. Moreover, the density of population is negatively correlated with the true rate of natural increase (ρ) as the following coefficient indicates:

$r_{\rho l} = -.6201 \pm .1560$

That is to say, the true natural increase is comparatively slower in densely crowded districts.

AGE CONSTITUTION

One of the most complicated situations involved in population growth is age constitution. Biologically it has direct concern with birth- and death-rates and industrial efficiency depends upon it. Since births and deaths, on one side, and industry, on the other side, are the most fundamental factors which influence the growth of population, the age constitution has naturally very intimate relationship with rapidity of population growth.

At first it was thought that it might be convenient to use a single numerical index of age constitution for the purpose of ascertaining the relationship. Accordingly, applying a formula suggested by Pearl,[9] the index of age distribution of each state was computed, but the correlation between the indices thus computed and the growth-rates of population turned out to be practically insignificant. However, from this result it cannot be concluded that the age constitution is unrelated to the growth-rate of population, because certain of the states have very similar age indices in spite of having quite different age distributions. Therefore the correlation coefficients between the growth-rates (b) and the proportion (percentages) of the population in the following six age groups were computed:

1. —Under 5 years
2. —5-14 years
3. —15-24 years

[9] Pearl, R. On a single numerical index of the age distribution of a population. *Proc. Nat. Acad. Sci.*, vol. VI, No. 7, July, 1920.

4. —25-44 years
5. —45-54 years
6. —55 years and over.

The results are of some interest from the regularity of the relationship which they exhibit:

(1) Under 5 years the coefficients are as follows:

$r_{b_1} = +.3328 \pm .0904$ 1920
$r_{b_1} = +.0690 \pm .1012$ 1910
$r_{b_1} = +.1925 \pm .0979$ 1900

Here we have positive correlation, though the coefficients cannot be considered significant.

(2) 5-14 years and
(3) 15-24 years:

$r_{b_2} = +.1123 \pm .1004$ 1920
$r_{b_2} = -.0965 \pm .1007$ 1910
$r_{b_2} = +.1236 \pm .1001$ 1900

$r_{b_3} = -.3054 \pm .0922$ 1920
$r_{b_3} = +.2092 \pm .0972$ 1910
$r_{b_3} = +.1573 \pm .0992$ 1900

Apparently the proportions of the population in these age groups have little if any correlation with the growth-rate.

(4) 25-44 years:

$r_{b_4} = +.4717 \pm .0791$ 1920
$r_{b_4} = +.5257 \pm .0736$ 1910
$r_{b_4} = +.6121 \pm .0636$ 1900

For this group of adults we have a definitely significant positive correlation with the population growth. From this we can say with certainty that in districts growing more rapidly in population there are relatively more adults in the age group 25-44. This result can be explained by the fact previously brought out, that states growing rapidly in population have relatively more immigrants from other states and foreign countries, these immigrants being mostly young or middle-aged persons. This immigration causes an excess of people in the ages 25-44, hence the positive correlation between the proportion of the population in this age class and the rate of growth of the population. The distortion in the proportion of the population in this age class due to migration affects the rate of growth in another way, namely through the fact that it tends to produce a relatively high birth-rate and a low death-rate.

(5) 45-54 years:

$r_{b_5} = -.1089 \pm .1005$ 1920
$r_{b_5} = +.0165 \pm .1016$ 1910
$r_{b_5} = -.0932 \pm .1008$ 1900

Here the correlation becomes again insignificant. The reason is evident if we proceed further to the next age group.

(6) 55 years and over:

$r_{b_6} = -.5391 \pm .0721$ 1920
$r_{b_6} = -.6188 \pm .0627$ 1910
$r_{b_6} = -.5493 \pm .0710$ 1900

These coefficients are negative and definitely significant, and indicate that there are comparatively more old people in states growing slowly in population. Proceeding from the group of middle age (having positive correlation with b) to the group of old age (having negative correlation with b), we pass through the intermediate group of middle-old age (45-54 years) which has no correlation with b. The death-rates tend to be higher in these states where the b's are smaller, on account of the relative abundance of old persons.

In Japan, if all prefectures are taken into account, the relationship between age constitution and the growth-rate of population is essentially similar to that of the United States. As a general rule, prefectures growing more rapidly in population contain relatively more middle-aged adults and fewer old persons. But if the six urban prefectures growing with extraordinary rapidity are eliminated, the correlation between b and the proportion of children under ten years of age in the population is closer than that of b and the proportion of middle-aged adults. The correlation coefficients are as follows:

Age Groups	*All Japan*	*40 rural prefectures*
0–9	$-.3264 \pm .0898$	$+.5135 \pm .0800$
10–19	$+.5334 \pm .0719$	$+.3075 \pm .0985$
20–44	$+.8094 \pm .0347$	$+.3307 \pm .0962$
45–64	$-.7020 \pm .0510$	$-.6228 \pm .0637$
65 and over	$-.6689 \pm .0556$	$-.5648 \pm .0735$

From these results and the following table comparing the age constitution of rural and urban districts, it can easily be perceived that youths are moving from the rural to the urban districts.

Age Groups	Age Distribution	
	Six urban prefectures	40 rural prefectures
0–9	22.54%	26.41%
10–19	22.07%	20.81%
20–44	38.02%	31.58%
45–64	13.50%	15.74%
65 and over	3.87%	5.46%
	100.00%	100.00%

The age constitution has also some definite relationship to the true rate of natural increase. For the United States, the correlation coefficients are as follows:

Age Groups	Sex	Coefficients of correlation between true rate of natural increase (ρ) and age distribution
0–14	both	$+.9033 \pm .1561$
20–49	males	$-.7965 \pm .1561$
15–44	females	$-.8454 \pm .1561$
50 and over	males	$-.8253 \pm .1561$
50 and over	females	$-.8484 \pm .1561$

The coefficients are all significant. The positive correlation between ρ and the proportion of children under fourteen years of age and the negative correlation between ρ and the proportion of persons above fifty years of age might be expected. But why should the proportion of middle-aged people, the groups which have as a rule high positive correlation with actual growth-rates of population, be negatively correlated with ρ? The above result seems paradoxical. But somewhat minute examination of the data reveals the fact that ρ is most intimately correlated with the percentages of children, and in the states where there are relatively more children, the percentages of other age groups are accordingly lower. Since such states have comparatively higher values of ρ, the correlation between ρ and the percentages of middle-aged adults is necessarily negative.

SEX RATIO—MARITAL CONDITION

These factors may have no direct influence on population growth, but may be regarded rather as social phenomena concomitant to the geographical differences of growth-rates of population. There exists, however, some social biological significance in the correlation between the growth-rate (b) and these factors. Here we denote by

s, sex ratio or number of male population per 100 females,

M, the percentage of males in marital condition, and
F, the percentage of females in marital condition.

The correlation coefficients between these factors and b are as follows:

In the United States:

$r_{bs} = +.6797 \pm .0547$ 1920
$r_{bs} = +.7820 \pm .0395$ 1910
$r_{bs} = +.7955 \pm .0373$ 1900

In Japan:

$r_{bs} = +.7528 \pm .0426$ . . . . . all Japan, 1925.

All these coefficients are positive and highly significant. From this result we know that in the United States as well as in Japan the excess of males over females is relatively higher in districts increasing more rapidly in population. This situation is obviously due to the fact that immigrants, not only foreign-born but also native, consist more of males than of females.

The correlation coefficients between b and M are

In the United States:

$r_{bM} = -.6502 \pm .0587$ 1920
$r_{bM} = -.7797 \pm .0399$ 1910

In Japan:

$r_{bM} = -.3309 \pm .0877$ 1925

These coefficients are all negative and significant. This result shows that there are fewer married males in districts increasing more rapidly in population, owing partly to the shortage of females.

The correlation coefficients between b and F are:

In the United States:

$r_{bF} = +.5600 \pm .0698$ 1920
$r_{bF} = +.5324 \pm .0729$ 1910

These coefficients are positive, contrary to the case with males. This result can be explained by the fact that the fewer the females relative to the males, the greater their opportunity to enter the marital state.

In Japan this sort of correlation coefficient is entirely insignificant.

SUMMARY AND CONCLUSIONS

The aim of this investigation is to examine the relationship between the rate of population growth and socio-biological factors which are concerned with it. For this purpose, the growth-rate of population (which will be denoted by b in the following discussion) for each district (state in the United States and prefecture in Japan) was determined on the

assumption that population growth may be considered geometrical in character if the time period is not too long. The correlation between the growth-rates thus computed and the various socio-biological factors which are subject to statistical treatment was studied in terms of the correlation coefficient. The results thus obtained are compared for Japan and the United States.

Furthermore, the true rate of natural increase (which will be denoted by ρ in the following discussion) was computed by the method suggested by Dublin and Lotka for each registration state in the United States using the data of the year 1920, and its correlation with the vital factors was studied.

The results which have become clear by this investigation can be summarized as follows:

1. The *growth-rate* of population is not uniform all over the country. In the United States the Mountain and Pacific regions have larger b's than the rest of the country. In Japan, the urban prefectures are increasing more rapidly in population than the rural.

2. The *birth-rate* (per 1000 population) is, generally speaking, higher in districts growing rapidly in population, except in the urban districts in Japan where the growth-rate is very high and the birth-rate very low.

If the crude birth-rate is corrected with respect to sex and reproductive period (birth-rate per 1000 married females of age 15-44 years), the correlation between birth-rate and b becomes insignificant in the United States, but is still significant in Japan, owing to certain social factors such as wealth, education, density, etc.

This birth-rate is very closely correlated with ρ in the positive sense.

3. The *death-rate* (per 1000 population) is negatively correlated with b. This correlation is not due to the geographical difference of sanitary conditions but to the age constitution of population. Therefore, the death-rate adjusted for age distribution has no correlation with b, either in the United States or in Japan.

4. The *infant mortality* rate is negatively correlated with b in the United States, owing partly to the geographical difference of economic conditions. In Japan there is no such relationship.

5. *Vital index* and excess of births over deaths are highly correlated with b as well as with ρ, both in the United States and Japan.

6. *Migration.* In the United States the percentage of foreign-born population is positively correlated with b, and negatively with ρ. The

reason for the former needs no explanation; the latter result is due to the tendency of immigrants to gather more in the urban districts than in the rural.

Immigration to a specified district from other parts of the country is positively correlated with b in the United States, but not in Japan, if six urban prefectures are eliminated.

Emigration to other districts is negatively correlated with b, both in the United States and in Japan.

In the United States the growth-rate of population born in the state of residence shows the highest correlation with b, and that of population born in other states and living in the specified state is next in order. The growth-rate of foreign-born population shows the smallest association with b.

In Japan, if the six urban prefectures are eliminated, the rate of population growth of each rural prefecture is not influenced significantly by the immigrants from other prefectures.

7. *Age constitution.* Generally speaking, in districts increasing rapidly in population there are relatively more middle-aged adults and fewer old persons, both in the United States and in Japan. This age constitution is obviously caused by intra- and international migration, and is the fundamental factor affecting the birth- and death-rates.

ρ has high positive correlation with the percentage of children in the population, but is negatively correlated with the percentages of adults and old people.

8. The proportion of population in rural and urban districts has no correlation with b, but is significantly correlated with ρ. The same relationship holds for the percentage of farmers in the total population.

9. *Density of population* has no correlation with b in the United States or Japan, but is negatively correlated with b in the agricultural states of the United States which have more than half of their total population in rural districts. It is negatively correlated with birth-rate and ρ, but is uncorrelated with mortality rate.

10. *Sex ratio—marital condition.* In the United States in districts where the b's are relatively larger, the excess of males over females is also larger, and consequently there are more unmarried males and fewer unmarried females. Such a condition is evidently caused by the migration of population. In Japan, the situation is similar to that of the United States, except that the proportion of females in the marital state has no correlation with b.

APPENDIX

TABLE I

STATE	GROWTH RATE b	TRUE RATE OF NATURAL INCREASE, ρ PER 1000 FEMALE POPULATION	GROWTH RATE OF Foreign-born population b_3	Population born in other states b_2	Population born in state of residence b_1
Maine	.0414	....	.1545	.2116	.0166
New Hampshire	.0682	....	.1645	.1177	.0154
Vermont	.0161	....	.0292	.0949	—.0007
Massachusetts	.1996	2.87	.2273	.1614	.1239
Rhode Island	.2100	....	.2245	.1893	.1911
Connecticut	.1904	6.12	.2722	.2307	.1584
New York	.1787	0.37	.2252	.2475	.1593
New Jersey	.2551	....	.3113	.3415	.2144
Pennsylvania	.1844	7.75	.2291	.2210	.1653
Ohio	.1478	2.35	.1352	.1918	.1340
Indiana	.1101	4.21	.0182	.0463	.1209
Illinois	.1895	....	.1817	.1011	.2183
Michigan	.2192	7.88	.1352	.0921	.2484
Wisconsin	.1858	5.46	.0025	.0891	.2457
Minnesota	.2853	6.59	.1347	.2086	.3756
Iowa	.0935	....	—.0463	—.0396	.1927
Missouri	.0776	....	—.0269	.0405	.1554
Dakota	.3076	....	.3240	.2901	.5151
Kansas	.1317	5.70	—.0071	—.0007	.1955
Delaware	.1128	....	.1768	.1890	.0649
Maryland	.1200	6.44	.0546	.1370	.0937
Dist. of Columbia	.2296	....	.1361	.2593	.1485
Virginia	.1066	14.20	.1167	.3935	.0811
West Virginia	.2379	....	.3320	.0946	.2551
North Carolina	.1679	17.91	.1826	.2963	.1441
South Carolina	.1331	12.74	—.0325	.3125	.1246
Georgia	.1782	....	.1142	.1801	.1586
Florida	.3309	....	.3976	.3562	.2943
Kentucky	.0974	10.21	—.1699	.0686	.1073
Tennessee	.1043	....	—.0203	.1052	.1045
Alabama	.1586	....	.1434	.0359	.1794
Mississippi	.1250	....	.0028	—.0688	.1612
Arkansas	.1292	....	.0801	.0882	.2484
Louisiana	.1605	....	—.0249	.0960	.1895
Texas	.2450	....	.2768	.1179	.3357
Montana	.4472	....	.2505	.4663	.6696
Idaho	.5452	....	.3092	.5651	.6654
Wyoming	.3857	....	.2243	.3838	.5752
Colorado	.2857	....	.1400	.2418	.4587
New Mexico	.2947	....	.3452	.5697	.2017
Arizona	.4502	....	.4557	.5562	.6282
Utah	.2490	19.11	.0530	.2360	.3182
Washington	.4794	1.64	.4076	.4320	.6491
Oregon	.3194	—0.02	.2434	.3440	.3231
California	.3511	—0.31	.2648	.4575	.3254

TABLE 2

PREFECTURE	RATE OF POPULATION INCREASE PER ANNUM PER 1000 PERSONS *b*	EXCESS OF BIRTHS OVER DEATHS PER 1000 PERSONS; AVERAGE FOR 1920-1922	1925	VITAL INDEX AVERAGE; 1923-1925
Hokkaido	11.57	19.8	20.1	206
Aomori	14.69	20.2	22.0	184
Iwate	12.79	15.8	18.5	173
Miyagi	16.13	17.0	20.8	190
Akita	8.29	16.3	19.4	171
Yamagata	11.77	15.5	18.6	181
Fukushima	10.75	14.3	18.2	181
Ibaragi	8.55	12.5	14.1	161
Tochigi	8.26	14.7	18.4	186
Gumma	12.27	14.3	16.0	170
Saitama	11.12	12.1	14.9	160
Chiba	9.28	12.8	13.7	146
Tokyo	39.41	8.8	...	...
Kanagawa	13.74	11.1	...	...
Niigata	8.12	12.6	15.6	161
Toyama	6.81	12.3	15.7	148
Ishikawa	0.94	8.2	10.4	130
Fukui	—0.30	9.6	11.0	139
Yamanashi	5.84	12.8	17.7	180
Magano	8.37	12.0	14.5	172
Gifu	11.34	14.1	15.7	166
Shisuoka	15.12	15.3	18.7	186
Aichi	21.07	11.4	...	...
Miye	7.08	11.2	12.7	157
Shiga	3.47	7.9	10.0	142
Kyoto	17.86	6.4	...	...
Osaka	34.06	5.5	...	...
Hyogo	12.94	9.3	...	...
Nara	6.71	11.2	11.6	158
Wakayama	9.70	12.0	12.6	168
Tottori	7.62	11.8	13.2	158
Shimane	2.16	8.3	10.5	134
Okayama	3.37	8.8	11.6	148
Hiroshima	9.63	11.5	13.2	152
Yamaguchi	10.07	8.4	11.9	145
Tokushima	5.82	10.0	13.3	152
Kagawa	6.54	14.1	15.2	164
Ehime	9.31	13.0	15.8	172
Kochi	4.90	9.6	12.3	155
Fukuoka	10.17	8.2	13.1	159
Saga	2.03	11.8	16.8	170
Nagasaki	4.87	11.5	15.5	178
Kumamoto	10.00	10.7	14.8	170
Oita	12.43	10.0	15.0	162
Miyazaki	11.86	14.6	17.3	188
Kagoshima	7.89	14.2	16.8	184
Okinawa	—4.92	7.8	6.8	148

DIFFERENTIAL MORTALITY IN THE AMERICAN NEGRO

(*Concluded*)

BY S. J. HOLMES

THE RESPIRATORY INFECTIONS

Ever since the period of emancipation our colored population has suffered from a very high death rate from respiratory diseases. It is not possible to trace with any accuracy the course of the death rate from specific respiratory affections in either whites or negroes owing to changes in the categories of diagnosis and the lack of adequate records. According to the U. S. Census of 1890 the pneumonia death rate in the Registration Area was 182 per 100,000 for whites and 278.97 for the colored population. In 1900 the pneumonia death rate was 184.8 for all whites and 349.0 for the colored. The rates for the whites in both 1890 and 1900 were raised somewhat on account of the high mortality from pneumonia among the foreign born which was 223.9 and 209.8 for these years, while that of the native born was 167 (1890) and 176 (1900). The rate for cities in the Registration Area for 1900 was 201.8 for whites and 263.1 for the colored, while in the rural parts of the area the rates were 135.3 and 176.7. In Baltimore the rates for all the pneumonias were over twice as high for the colored population as for the whites in every year between 1906 and 1924 with the exception of the year of the great epidemic of influenza in 1918. Deaths from pneumonia are notoriously more common in cities than in rural districts, and the fact that the pneumonia rate was higher in both races in 1900 than in 1890 may be due to the increasing urbanization of our population.

The lack of sufficiently complete data and the fact that the pneumonia death rate fluctuates so greatly owing to climatic changes and the varying prevalence of other intercurrent diseases such as measles and influenza which dispose people to contract pneumonia make it difficult to ascertain whether pneumonia was actually increasing or decreasing previous to 1900. The data from Baltimore and other cities which kept records during this time do not show any consistent trend. Since

1900 the general death rate from pneumonia has undergone a gradual decline. Not only is this true for the Registration Area as a whole, but also for its various subdivisions. It is possible that a part of this decline is spurious since, as stated in the volume on Mortality Statistics for 1914, "the practice of writing merely 'Pneumonia' or 'Broncho-pneumonia' and of omitting the name of the disease that was the primary cause was far more prevalent than it is today." The great epidemic of influenza in 1918, with the slower rise of the pneumonia rate in the two years preceding it, forms a striking interruption of the general decline of the curve of pneumonia mortality. The pneumonia rate after 1918 has been very uneven owing largely to less severe epidemics of influenza, but in general the rates after 1918 have shown a downward course. Taking for comparison the years 1910-1915 and 1920-1925, thus omitting the years of the great epidemic, we find a marked decrease in the pneumonia death rate in the latter period. I have calculated the death rates for total pneumonia in the Registration States of 1920 for the period 1920 to 1924 inclusive, but these show such extensive annual fluctuations that no conclusions can be drawn from data covering so short a time. In the Registration States of 1920 for this whole five year period in question the death rate for all respiratory diseases was 120.85 for the whites and 199.05 for the colored. Similar rates for respiratory diseases are shown by the wage earners insured by the Metropolitan Life Insurance Company (1911-1916), namely 124.38 for the whites, and 190.53 for the colored. The fact that the Metropolitan group consisted of wage earners probably explains the smaller difference in the death rates of the two races. From the data published from time to time by the Metropolitan Life Insurance Company one would conclude that among white industrial policy holders there has been a noticeable fall in the pneumonia death rate between 1911-1916 and the last quinquennium among the whites, and an actual rise among the colored. This is true for respiratory diseases as a whole and for pneumonia lobar and undefined.

The negro seems peculiarly liable to succumb to lobar pneumonia as contrasted with broncho-pneumonia. While the death rate from broncho-pneumonia has been greater among the negroes than among the whites the ratio of deaths (white to colored) from this cause in the Registration States of 1920 (1920-1924), namely 7.81, was higher than it was for deaths from all causes, 7.607, whereas for lobar pneumonia the corresponding ratio was 6.69. That the ratio 3.01 for "pneumonia undefined"

TABLE 14

Death Rates from Pneumonia (all forms) per 100,000, White and Colored in Selected States and Cities 1920-1927

		1920	1921	1922	1923	1924	1925	1926	1927
Louisiana	W	92.8	59.4	65.5	71.1	82.1	73.9	74.2	55.0
	C	132.3	89.5	109.5	124.5	149.9	145.0	140.8	128.6
Maryland	W	133.2	92.0	97.3	129.5	111.6	107.8	121.4	99.7
	C	252.5	179.9	192.7	274.3	249.4	288.6	284.3	268.0
Mississippi	W	81.5	58.8	66.9	64.5	77.6	65.8	79.2	57.0
	C	122.4	72.5	87.4	90.1	98.1	91.0	105.9	85.8
North Carolina	W	87.4	66.5	76.2	76.2	86.6	72.5	78.0	59.8
	C	135.4	94.4	115.5	125.2	141.8	117.8	131.6	113.6
Tennessee	W	100.5	66.0	75.9	81.2	84.2	67.3	89.7	71.3
	C	187.5	110.8	138.0	172.9	188.3	170.6	183.9	149.9
Virginia	W	81.7	69.3	67.8	74.3	72.0	59.3	68.6	53.8
	C	154.7	115.5	120.8	133.3	140.2	125.2	133.6	118.5
Atlanta	W	114.0	76.2	77.1	99.7	...	...	74.1	...
	C	362.9	194.0	274.0	367.5	...	...	256.6	...
Baltimore	W	160.0	110.0	113.5	148.0	133.5	133.6	144.2	...
	C	327.9	221.5	266.3	377.4	375.7	453.5	408.4	...
Birmingham	W	120.9	82.3	76.7	94.1	127.9	114.4	111.8	...
	C	290.7	205.2	194.6	280.9	351.5	318.5	286.6	...
Dallas	W	102.3	64.7	72.6	72.1	81.4	89.3	95.3	...
	C	214.8	122.9	247.3	215.2	205.8	283.5	243.4	...
Indianapolis	W	157.8	74.1	106.8	105.2	98.6	100.7	101.8	...
	C	344.2	195.8	209.9	283.9	255.0	208.0	255.2	...
Memphis	W	147.0	79.8	91.6	140.0	110.2	98.5	118.0	...
	C	347.0	146.0	217.9	282.2	337.5	311.2	242.5	...
Nashville	W	138.3	87.4	103.6	130.1	119.6	90.6	140.8	...
	C	283.8	198.7	175.2	279.8	218.8	203.1	317.9	...
New Orleans	W	127.2	82.3	87.0	100.0	85.5	82.8	100.8	...
	C	350.3	210.5	236.8	265.4	318.7	329.6	319.9	...
Richmond	W	100.2	61.8	71.1	66.5	65.2	56.4	77.0	...
	C	363.5	253.8	269.4	252.0	287.5	263.3	301.4	...
Washington, D. C.	W	128.2	69.1	79.5	109.9	75.1	60.2	84.2	...
	C	352.4	242.1	253.8	392.6	255.0	209.7	266.5	...

Rates for cities in 1927 were not calculated on account of uncertainties in the estimates of populations.

in the same period and area shows a very high negro mortality is doubtless due to less precision in specifying the causes of negro deaths. That a large part of these deaths was doubtless due to lobar pneumonia is made evident by the result of an investigation of the accuracy of reports on the cause of deaths carried out by the Bureau of the Census in 1911. Further inquiry into reports of deaths certified as due to pneumonia not otherwise defined "resulted in a change of assignment in 416 out of 1,013 cases (41.1 per cent), and confirmed the diagnosis of lobar pneumonia in 518 cases (51.1 per cent) leaving only 79 deaths still undefined."

For some reason the racial distribution of deaths from pneumonia in the records from Baltimore compiled by Howard does not correspond closely with the preceding data. For the period 1906-1920 the death rates of whites and colored were 69 and 178 (ratio 1 : 2.57) respectively, while those for lobar pneumonia were 110 and 265 (ratio 1 : 2.40). Since broncho-pneumonia is predominantly a disease of childhood while lobar pneumonia is relatively more frequent among adults, differences in age composition of the two races in different localities would give rise to differences in the relative prevalence of the two affections. This, however, would hardly account for the distribution of the pneumonias in Baltimore, nor for the marked variations in the proportions of deaths due to these two causes in different cities. Aside from the uncertainties of diagnosis which are considerable there are also differences in the prevailing types of infection in different times and places. The preponderance of data indicates, however, that the negro is relatively susceptible to the lobar type of pneumonia both in infancy and in more advanced ages.

Instructive data on the prevalence of pneumonia in the two races are furnished by the records of white and colored recruits for the late war. "For 1917," according to Love and Davenport ('19), "the rate for lobar pneumonia was four and a half times, for broncho-pneumonia three times, as great for colored as for white men. In the ten year record the colored rate is 2.2 times the white rate for broncho-pneumonia. Given, then, troops under similar environment in the presence of conditions favoring pneumonia, there are relatively two to three times as many colored troops as white who show symptoms of the disease." During the great war the admission rate for broncho-pneumonia was a little over twice as high for the colored as for the white soldiers while for lobar pneumonia the admission rate was over four times as high for the colored. The death rates for these diseases were 1.80, whites,

and 3.71 colored, for broncho-pneumonia, and 1.96, whites, and 7.75, colored, for lobar pneumonia. Even among respiratory diseases lobar pneumonia stands out as a very prevalent and fatal disease in the colored soldiers as in the colored civil population. The statistics for respiratory diseases for the U. S. Army during the entire period of the war are given in the following table:

TABLE 15

Admissions and Deaths, Numbers and Rates per 1,000, in the U. S. Army, April 1, 1917 to December 31, 1919

		Influenza	Bronchitis	Broncho-pneumonia	Lobar pneumonia	Total
TOTAL ENLISTED TROOPS						
Admissions, Number	W	671,322	214,561	24,422	31,903	942,208
	C	59,448	20,045	4,825	11,482	95,800
Admissions, Rate	W	186.50	59.61	6.78	8.86	261.75
	C	207.46	69.95	16.84	40.07	334.32
Deaths, Number	W	20,888	334	6,480	7,073	34,775
	C	2,287	42	1,063	2,222	5,614
Deaths, Rate	W	5.80	.09	1.80	1.96	9.75
	C	7.98	.15	3.71	7.75	19.59
U. S. ARMY IN THE U. S.						
Admissions, Number	W	476,816	.....	13,297	21,886	660,400
	C	38,863	.....	2,759	7,016	61,601
Admissions, Rate	W	242.62	.....	6.77	11.14	336.04
	C	266.51	.....	18.92	48.10	422.41
Deaths, Number	W	14,617	.....	3,429	4,330	22,400
	C	1,567	.....	634	1,363	3,567
Deaths, Rate	W	7.44	.....	1.74	2.20	11.39
	C	10.74	.....	4.35	9.35	24.46
U. S. ARMY IN EUROPE						
Admissions, Number	W	176,240	.....	10,761	9,000	256,099
	C	18,619	.....	1,986	4,149	31,435
Admissions, Rate	W	119.92	.....	7.32	6.12	174.25
	C	152.10	.....	16.32	33.89	256.89
Deaths, Number	W	5,753	.....	2,919	2,414	11,390
	C	628	.....	395	778	1,839
Deaths, Rate	W	3.91	.....	1.99	1.64	7.75
	C	5.15	.....	3.23	6.36	15.05

As may be seen from the table the death rates for all forms of respiratory infections are higher for the colored troops but they are very much higher for lobar pneumonia than for other forms of respiratory infections.

The Army data are of especial interest in relation to the problem of racial susceptibility to respiratory disease. Unquestionably a considerable part of the high mortality among our colored civil population is to be attributed to unfavorable conditions of life. But during the great war white and negro recruits were given much the same medical examination when they entered the Army and they all had to pass much the same tests of physical fitness. While in the Army they lived under more or less similar conditions; nevertheless the pneumonia rates among the colored were even higher relative to those of the whites than they are in the general population. Even in the Army conditions may have been somewhat less favorable for the negroes, but this fact surely cannot account for the enormous differences in the incidence of pneumonia, and especially lobar pneumonia, in the two races. These same differences are found both in the soldiers quartered in America and in those who were in Europe.

In dealing with the problem of racial susceptibility it may be instructive to allude briefly to the death rates from pneumonia among different elements of the white population. The foreign born inhabitants show a distinctly higher pneumonia rate than the native born.

TABLE 16

Pneumonia Mortality According to Race and Country of Birth.
Rates per 100,000

DATE	ALL WHITES	NATIVE BORN WHITES	FOREIGN BORN WHITES	COLORED
1890	182.24	167	223.9	278.97
1900	184.8	176	209.8	349.00

As shown in the table the mortality of the foreign born is intermediate between that of the native born and the colored population. The use of adjusted instead of crude death rates reduces the rate of people whose mothers were born abroad while raising the rate of the native American stock of native parentage, but the differences are nevertheless sufficiently striking. Among the foreign born there are striking differences among white immigrant groups. The Irish have an exceptionally high death rate from pneumonia as they have from tuberculosis. The pneumonia death rate among the Italians is also

high, although their mortality from tuberculosis is less than that of most immigrant stocks. The marked differences in the pneumonia death rates of different groups of the white race have been adduced as an argument that the high rate among negroes is due almost entirely to environment instead of racial heredity. The argument is based on the assumption that the differences in mortality among the whites are the result of environmental causes. If one compares the pneumonia rates obtaining in different countries with the rates occurring in the representatives of these countries in America there will be found to be no very close correspondence. This does not necessarily disprove the existence of racial diatheses provided we admit that environmental factors are, as they are known to be, of great importance also. At present we cannot be said to have conclusive evidence either for or against the existence of racial diatheses to pneumonia in the subdivisions of the white race.

There are differences in the age incidence of pneumonia characteristic of the two races which are similar to those of tuberculosis. In early life (1-4 years) the death rate from lobar pneumonia in negroes is over twice that of the whites. From five to fifteen the racial differences in mortality are reduced, but from fifteen to twenty-five the rate for the colored population is over three times that of the whites. The trend for the age groups is well shown in the data from the wage earners insured by the Metropolitan Life Insurance Company. Racial differences in the age distribution of bronchopneumonia as shown by the data of the company referred to are also striking.

Mention should be made of the sex distribution of lobar pneumonia because for some age groups, especially in ages 25-55, according to the Metropolitan experience, the death rate of males is about twice that of females. Previously and subsequently to this period the preponderance of male deaths is much less. The age of the highest death rates for males as compared with females corresponds roughly with the period of greatest difference in exposure of the two sexes. For bronchopneumonia the sex differences in mortality are very much less for nearly all age groups and frequently are to the advantage of the males. The experience of the Metropolitan as to sex differences of mortality from the two types of pneumonia 1911-1916, may be summarized as follows:

	White		*Colored*	
	Male	Female	Male	Female
Lobar pneumonia	82.6	63.0	141.5	97.2
Bronchopneumonia	29.6	29.2	37.7	33.7

One very significant fact in connection with the possible influence of racial heredity upon mortality from pneumonia is that the case fatality from this disease seems to be actually lower among the negroes than among the whites. In this respect pneumonia stands in sharp contrast to tuberculosis, whose case fatality is very much higher in the colored race. If unfavorable environment and inadequate care were mainly responsible for the fatal termination of cases of pneumonia we should certainly expect that the negroes would show a smaller percentage of recoveries than the whites. Dr. F. B. Kelly ('26) in describing a series of 6,500 cases of lobar pneumonia in the Cook County Hospital, Chicago, states that "From 1917-24 the case fatality was 39.2% for 3,749 white males and 34.7% for 818 white females, 30.6% for 1,876 colored males and 31.9% for 388 colored females. Every year the case fatality in the male negroes has been 5 to 13 per cent below that of the male whites, with a difference of 8.8% for the entire 7½ years. The female negro case fatality has been 4.6% less than that of white females in the same period. In two years, however, the difference favored the white females." The same writer states that "Chittard found a case fatality of 31.2% in 250 cases compared with 30.6% in the native born whites." In commenting on the relation of case fatality to age Dr. Kelly states that "The case fatality for the two races was the same up to the '40 year and over' groups, above which the negroes had the greater resistance."

It is somewhat dangerous to draw conclusions on the basis of human material which comes into a large city hospital because this material may be selected in quite different ways from among the general population of the two races. The writer states that "it may be claimed that the recent migration northward has brought a larger percentage of the strong hardy members of the race with a naturally greater resistance than general. However, most of these newcomers are under 40 years of age, in which class the case fatality has been the same as in the white race."

In order possibly to secure more information on this subject than is available in the literature, I have written to several physicians associated with large hospitals in the Southern States. Most of the replies failed to supply any conclusive data. Dr. J. H. Musser, however, who kindly furnished me with some statistics from the Charity Hospital in New Orleans, reported that in 1926, out of 101 cases of lobar pneumonia among the negroes, 33, or 32.7 per cent died, while out of 65 cases among the whites, 30, or 46.2 per cent died. In 1929 the case mortality was slightly higher in the negroes. Dr. Musser states that

while the negroes are especially prone to contract pneumonia, "we have never considered that after the negro has developed pneumonia he is more likely to die than the white man."

A much larger material collected under more nearly comparable conditions is furnished by the experience of the U. S. Army. Howard and Love in summarizing the data on mortality from pneumonia in 1918 give the following picture: "For the white troops the death rate for these two pneumonias was 4.89, and for the colored 15.81. The total number of pneumonia cases among the white troops in the United States and Europe, counting the cases complicating measles and influenza, was 94,505 with 28,969 deaths. Among the colored troops, enlisted men, there were 19,319 cases with 4,720 deaths. The incidence rate for the white troops was 44.11 and for the colored, 115.28. The death rate for the white was 13.52, and for the colored, 28.17. The case mortality for primary and secondary pneumonia for the white was 30.6 per cent, and for the colored 24.4 per cent; for primary pneumonia for the whites it was 26.4 and for the colored 21.6 per cent. It is apparent, then, that the colored race has an incidence rate nearly three times as high as that for the white, a death rate nearly twice as high, but a case mortality rate about 20 per cent lower for all pneumonia, both primary and secondary." The case fatality rates for the whole period of the war are as follows:

TABLE 17

Percentage of Deaths to Cases of Respiratory Diseases in the American Army, 1917-1919

	INFLUENZA		BRONCHO-PNEUMONIA		LOBAR PNEUMONIA	
	W	C	W	C	W	C
U. S. Army in U. S.	3.1	4.0	25.8	23.0	19.8	19.4
U. S. Army in Europe	3.3	3.4	27.1	19.9	26.8	18.8
Total U. S. Army	3.1	3.8	26.5	22.0	22.2	19.3

One explanation of the relatively low case mortality of the negroes is that pneumonia is more apt to attack negroes at an early and hence more vigorous age. If for corresponding age periods the negroes should exhibit a lower case mortality than the whites we should have the rather unusual combination of greater proneness to contract a disease with a greater power of resisting it when once contracted.

That negroes should have a relatively low case fatality from pneumonia is all the more remarkable because they suffer more than the

whites from malaria, tuberculosis, syphilis and other devitalizing afflictions which would naturally tend to reduce their power of recovery. Dr. Reitzel ('28) in studying the rôle of syphilis in the mortality from pneumonia in a hospital at Galveston, Texas, remarks that "Among the 146 colored patients of this series (all cases of lobar pneumonia) 50 had either positive Wassermanns or post mortem evidence of syphilis, and in this group the fatality was 28 per cent. There were 57 who had negative Wassermanns with a fatality of 10.5 per cent. Among the 39 cases in which the Wassermanns were not reported, the fatality was 25.6 per cent." The author remarks upon the delayed resolution of pneumonia in syphilitic patients and quotes the experience of other physicians to the same effect. In connection with what has been said concerning case fatality in the two races it may be mentioned that in the same hospital the case fatality of 143 whites for lobar pneumonia was 19.5 per cent, while that of the 146 colored was 21.6 per cent.

The negro death rate from bronchitis is considerably higher than that of the whites, but in infancy the ratio of negro to white deaths is less than that of deaths from all causes. For the whole period of the great war the admission rates for white and colored soldiers were 59.61 and 69.95 respectively, and the death rates .09 and .15 per 1,000.

INFLUENZA

Owing to the intimate association of influenza with various kinds of pneumonia it is practically impossible to obtain data on deaths from influenza free from complications with these other infections. Undoubtedly a large part of the higher influenza death rate in the general colored population is attributable to unfavorable conditions combined with a greater susceptibility to pneumonia. During the great war the difference in the death rates of white and colored troops was not nearly so great as it was for pneumonia, being 9.78 for the colored and 5.80 for the whites. There is quite satisfactory evidence that the negro is not attacked so readily as the whites, but that when once attacked his chances for recovery are not so good.

The data on morbidity from different camps during the great war are somewhat conflicting, but there are several causes which may account for this fact. Soldiers who had been seasoned by several weeks' service were not so liable to contract influenza as were new recruits. As Howard and Love observe, troops from the rural districts, especially in the south, were more susceptible to influenza and pneumonia than those from the northern and eastern states. Opie *et al.* ('19) found

that at Camp Pike the negroes who had been in camp for six weeks or more and who had furnished no previous cases of the infection developed influenza in only 7.6 per cent of cases, while in a new group in which influenza had appeared there were 43.6 per cent of cases. While Opie *et al.* attribute differences in influenza mortality to environmental factors instead of race, the attack rate of the "old" white soldiers was about double that of the seasoned negroes. For Camp Pike as a whole the rates were 246 per 1,000 for whites and 133 for negroes.

Although in a few camps the negroes were reported to have been attacked more frequently than the whites, in others the negroes had the lower rate. Howard and Love found that in 1918 the influenza rate for white troops of the south was 247.11 and for the colored 154.58. However, for the whole war period, April 1, 1917-December 31, 1919, there were 671,322 admissions for whites, or 186.50 per 1,000, and 59,448, or 207.46 per 1,000 for the colored. For the same period the admission rate for white soldiers was higher than it was for the colored in the army quartered in the United States for nearly every month, while for the army in Europe the admission rate was higher for the colored troops for every month except October, 1918, when the epidemic was at its height.

In surveys of the incidence of influenza among the civil population carried out by the U. S. Public Health Service soon after the epidemic of 1918 the attack rate was found to be higher in the whites. According to Frost ('20), "In the seven localities with considerable colored population, the incidence rates among the colored were uniformly lower than among the white, the differences persisting after adjustment of the rates to a uniform basis of sex and age distribution. . . . . This relatively low incidence in the colored race is contrary to what would have been expected *a priori* in view of the fact that the death rate from pneumonia and influenza is normally higher in the colored than in the white, and that the colored population live generally under conditions presumably more favorable to the spread of contact infections." When comparison was made between the case fatalities in these same localities the colored population was usually found to have the higher rate. In the U. S. Army experience there were 20,888 deaths attributed to influenza among the whites and 2,287 among the colored, giving a percentage (deaths to cases) of 3.1 for the whites and 3.8 for the colored.

In a study of the racial mortality from influenza in the 12,000,000 industrial policy holders of the Metropolitan Life Insurance Company, Frankel and Dublin ('19) find that in the first three months of the

epidemic, when the great outburst occurred, the death rate was 1,844 per 100,000 for white males and 1,522 for colored males, 1,723 for white females, and 1,504 for colored females. "After the severest period of the epidemic had passed, the colored group showed higher rates than the whites, and the amount of excess approximated what had prevailed in normal times, as the distance from the explosive period of the epidemic increased."

Craig and Dublin ('19) in their survey of the Metropolitan experience for 1918 state that "While the colored race in 1918 suffered higher mortality than the white, the absolute amount of excess among the negroes was very much less. Thus, while the maximum excess among white lives at the age period 25-29 years was 14 deaths per 1,000, that for the colored for the same age period was only 5.7 deaths per 1,000." During October, November and December, 1918, the death rate from lobar pneumonia, bronchopneumonia and influenza combined was higher in the white industrial policy holders; "this in spite of the fact that the colored race ordinarily has much higher death rates from pneumonia than the whites." Up to age 15 the colored population had the higher rates from the pneumonias and influenza, but for each quinquennium after age 15 the rate was less, the differences being most marked in the period between 20-40 years of age.

The U. S. Mortality Statistics show that for the whole Registration Area the negro death rate from influenza in 1918 was somewhat higher than that of the whites, but that the difference was very much less than in previous and subsequent years. In Virginia and in several cities with a large colored population, (Indianapolis, Kansas City, Washington, D. C., Memphis, Nashville, Louisville, Norfolk, and Richmond) the death rate from influenza was lower in negroes than in whites.

Normally influenza affects most severely the very young (1-4 years) and those past the middle period of life, but in the months when the epidemic was most severe there was a third peak in the curve of mortality between ages 20 and 40. This peak was not so high relatively in the negroes as in the whites, and as the epidemic continued it gradually disappeared until in the later period it was not manifested at all. Apparently both the age incidence and the race incidence of the disease changed as time went on. Whether this was due to changes in the persons attacked, changes in the causative organism, differences in the relative prevalence of the several other kinds of bacteria associated with the disease, or in part to the influence of climate is uncertain. Certainly influenza during the period of its greatest severity in the latter

part of 1918 behaved in several ways differently from the disease which usually goes under this name.

When we bear in mind that the negro is peculiarly apt to contract respiratory diseases, the data from the distribution of deaths during the latter part of 1918, together with what we know of the morbidity of the two races from this disease, indicate that the negro is less liable than the whites to become infected with the type of influenza which prevailed during the last great epidemic. This statement is not intended to apply to previous great waves, nor to what is commonly diagnosed as influenza during ordinary times.

The case mortality, to judge by the Army experience, both in the United States and in Europe, is a little higher for the negroes than the whites, but this is probably due to the negro's susceptibility to pneumonia. Possibly the negro may be less prone to contract influenza for the same reason that he is relatively free from colds and affections of the nose and sinuses, but we do not know whether influenza makes its first inroads in the nose, throat, or respiratory tract.

DISEASES OF THE NOSE AND THROAT

According to the Army Medical Statistics negroes are less affected by laryngitis, tonsillitis and pharyngitis than are the whites. The admission rates per thousand 1917 to 1919 were as follows:

	Whites	*Negroes*
Laryngitis	4.54	2.40
Tonsillitis, acute	43.97	31.25
Tonsillitis, chronic	6.00	2.14
Pharyngitis	12.87	9.02

In these affections it is not the ectoderm which is chiefly involved but the parts attacked are very often affected by inflammations which spread to them from the ectodermic lining of the vocal cavity and nose.

Our tentative conclusion in regard to the relation of the ectoderm to the infection naturally leads one to expect a relatively low prevalence of nasal infections in the negro race. The U. S. mortality statistics show a somewhat higher death rate from diseases of the nasal cavity among the negroes than among the whites. Many deaths attributed to "diseases of the nose" are doubtless due to syphilis, which may play havoc with the nasal cartilages and bone and finally involve the brain. Most other nasal troubles are rarely fatal so that our mortality statistics tell us

little in regard to the natural resistance of the nasal membranes to infection. Statistics of the relative prevalence of the common nasal infections in whites and negroes are not as extensive as could be desired, but so far as they go they indicate that the negroes are less afflicted than the whites. This is the testimony of Dr. Scheppergrell ('95), who has studied an extensive series from both races. Rhinitis, sinusitis and most other diseases of the nose and adnexa showed a much lower incidence among negroes than among white troops during the late war.

CANCER

The real influence of racial heredity upon the prevalence of cancer and other malignant tumors is difficult to ascertain on account of the fact that food habits, to say nothing of other environmental factors, have been found to exert a potent influence in the causation of these abnormal growths. Quite aside from the average lower age of primitive peoples and the fact that cancer is more apt to go unrecognized among them, there is no doubt that many racial groups are remarkably free from cancer. There is also considerable evidence that when peoples suffering little from cancer come to adopt the food habits of modern civilized mankind their mortality from cancer increases. It will not be necessary to discuss the varied incidence of cancer, since the subject has been thoroughly treated in Dr. F. L. Hoffman's treatise on *The Mortality from Cancer Throughout the World,* and in his later brochure on *Cancer in Native Races.*

What is known of the relation between habits of life and the prevalence of cancer naturally disposes one to attribute the rather striking differences in the cancer mortality of whites and negroes to causes other than race. Statistics on cancer are liable to be misinterpreted for a number of reasons. Since cancer is predominantly a disease of middle and later life the age composition of a population has a marked influence upon its prevalence. As the negro population has fewer persons in the higher age groups we should expect to find among them a lower cancer mortality than in the whites. Then cancer statistics are notoriously influenced greatly by the adequacy of diagnosis of the causes of death. Here again the statistics would tend to show a higher cancer mortality among the better circumstanced whites. Other things equal, urban rates for cancer exceed those of rural districts, and possibly climate may be, in part, responsible for the higher cancer rates in northern than in southern states.

If in order to get rid of the influence of age composition we compare the cancer mortality of negroes and whites in corresponding age groups we find that at every age the negroes have the lower mortality. This fact is not necessarily indicative of a true racial difference; it may be due to differences in habits, especially as regards food, and less accurate diagnoses of causes of death in the negro. The facts which are of the greatest significance in relation to problems of racial heredity are those concerning the parts of the body most frequently attacked in the two races. In order to get a large amount of data for comparisons I have summarized the statistics of cancer for the Registration Area of 1920 for the years 1920 to 1924 inclusive. For such a comparison taking a fixed area instead of one which is changing by the addition of new territory avoids a number of sources of error. I have calculated the cancer death rates of negroes and whites for various age groups and for several types of cancer. The results show consistent and very striking differences in the bodily distribution of cancer in the two races. Cancer of the stomach, liver, intestine, rectum and peritoneum is more prevalent in the whites than in the negroes for the several age groups, a fact which may be explained as due to differences in food or in accuracy of diagnosis. The same explanation might also be applied to the higher white rate for cancer of unspecified location. That inadequate diagnosis alone cannot account for the findings is indicated by the fact that the racial differences in the prevalence of the classes of cancer referred to consistently increase with age and as the cancer rates themselves increase. The same age trend is shown even more conspicuously in the easily recognized cancers of the skin. There seems to be no reason why errors in diagnosing cancer should be much greater in one age than in another.

Cancer of the female generative organs is more prevalent in negroes than in whites in every age group, but more especially in younger women. This predominance in the early age groups is sufficiently great to outweigh the relatively low negro rate for other kinds of cancer and to make the total cancer mortality of females in the child bearing period higher in the colored than in the white population. A large proportion of uterine cancers are traumatic in origin. They are notoriously rare in women who have not borne children. Less adequate care after childbirth, resulting in the neglect of lacerations and other injuries, would naturally tend to cause more uterine cancer among negro women. Among negroes it is probable that a smaller percentage of uterine cancers are recognized in their early stages and remedied by surgery.

But however one may interpret the data furnished by our mortality statistics, the proneness of negro women to develop fibroid uterine growths has often been remarked upon and probably represents a true racial peculiarity.[1] Apparently uterine cancer is much more rare in certain African tribes than in the American negro. Given, possibly as a result of changed habits of life, an increased proneness to develop cancer, the injuries due to child bearing may well account for an increased incidence of malignant uterine growths.

TABLE 18

Deaths and Death Rates per 100,000 of White and Colored Population from Cancer in Registration States of 1920 for 1920-1923 Inclusive

	TOTAL CANCER				BUCCAL CAVITY			
AGE	No. W.	No. C.	R. W.	R. C.	No. W.	No. C.	R. W.	R. C.
35-39	2,873	227	22.576	19.047	150	11	1.179	0.9229
	6,465	918	56.100	81.569	41	8	0.356	0.7108
40-44	4,837	384	46.634	43.223	322	33	3.105	3.7145
	10,689	1,258	109.620	151.85	64	7	0.6563	0.8449
45-49	7,753	516	79.323	53.61	566	30	5.791	3.117
	14,815	1,430	172.34	215.02	97	13	1.1284	1.956
50-54	12,137	682	150.09	103.49	867	40	10.722	6.0698
	18,576	1,353	254.159	277.53	127	11	1.737	2.258
55-59	15,689	613	252.12	159.05	1,054	37	16.939	9.6008
	20,148	1,085	352.72	376.18	144	16	2.5209	5.5476
60-64	19,613	612	376.41	188.17	1,281	29	24.586	8.9167
	22,245	982	467.789	395.97	175	14	3.680	5.6456
65-69	19,748	520	564.33	236.68	1,197	32	34.207	14.569
	20,650	776	623.09	444.78	216	11	6.517	6.3054
70-74	17,763	387	769.52	278.41	1,176	22	50.94	15.827
	18,047	607	777.47	475.54	187	11	8.056	8.6177
75-79	12,638	264	903.82	329.99	906	21	64.793	26.249
	13,455	329	898.59	442.22	195	10	13.029	13.442
80-84	6,290	118	1027.7	318.65	578	4	94.45	10.802
	7,591	206	1028.3	485.36	179	2	24.25	47.14
85	2,429	58	1078.9	366.65	265	9	117.76	56.899
	3,410	119	1155.6	626.23	117	6	39.68	31.576

[1] Dr. C. I. Miller in a survey of nearly 3,000 cases of fibroids of the uterus in the Charity Hospital, New Orleans, finds that an overwhelming proportion occurs in colored women and he estimates that fibroids develop in from one-third to one-half of colored women over 50 years of age.

TABLE 18—*Continued*

Deaths and Death Rates per 100,000 of White and Colored Population from Cancer in Registration States of 1920 for 1920-1923 Inclusive

AGE	STOMACH, LIVER				PERITONEUM, INTESTINE AND RECTUM			
	No. W.	No. C.	R. W.	R. C.	No. W.	No. C.	R. W.	R. C.
35-39	1,143	116	8.981	9.733	589	37	4.629	3.105
	1,056	151	9.165	13.416	765	84	6.638	7.464
40-44	2,172	202	20.941	22.737	851	46	8.205	5.178
	1,989	204	20.398	24.623	1,130	94	11.347	13.459
45-49	3,708	288	37.938	29.922	1,161	55	11.879	5.7143
	3,220	266	37.459	39.995	1,635	90	19.020	13.533
50-54	6,092	385	75.339	58.422	1,665	79	20.591	11.989
	4,916	300	67.261	61.536	2,338	81	31.989	16.614
55-59	8,045	342	129.28	88.727	2,209	74	35.499	19.198
	6,290	278	110.12	96.385	2,729	86	47.775	29.817
60-64	9,902	310	190.03	95.319	2,760	84	52.971	25.827
	7,975	287	167.71	115.72	3,261	69	68.575	27.826
65-69	9,939	288	284.02	131.08	2,724	50	77.843	22.758
	8,177	214	246.73	122.66	3,144	62	94.867	35.542
70-74	8,412	205	364.42	147.48	2,341	36	101.42	25.898
	7,750	171	333.87	133.97	2,817	56	121.36	43.872
75-79	5,570	131	398.35	163.75	1,674	27	119.72	33.749
	5,581	106	372.73	142.48	2,284	24	152.54	32.259
80-84	2,515	61	410.93	164.74	747	9	122.05	24.303
	2,984	59	404.25	139.03	1,164	16	157.69	37.699
85	786	22	349.19	139.07	226	5	100.40	31.608
	1,131	36	383.28	189.48	497	8	168.45	42.09

TABLE 18—*Continued*

Deaths and Death Rates per 100,000 of White and Colored Population from Cancer in Registration States of 1920 for 1920-1923 Inclusive

AGE	BREAST, FEMALES ONLY				FEMALE GENITAL ORGANS			
	No. W.	No. C.	R. W.	R. C.	No. W.	No. C.	R. W.	R. C.
35-39	1,227	113	10.647	10.039	2,672	499	23.186	44.335
40-44	2,272	167	23.300	20.158	4,060	684	41.637	82.560
45-49	3,194	222	37.157	33.380	5,074	735	59.027	110.51
50-54	3,533	212	48.339	43.486	5,505	628	75.320	128.81
55-59	3,558	168	62.288	58.247	5,087	433	89.056	150.12
60-64	3,331	140	70.047	56.456	4,851	381	102.01	153.63
65-69	2,572	106	77.607	60.757	3,994	306	120.52	175.44
70-74	2,128	106	91.674	83.044	2,837	189	122.22	148.07
75-79	1,630	60	108.89	80.647	1,869	101	124.83	135.76
80-84	1,032	33	139.81	77.771	900	68	121.93	160.23

TABLE 18—*Continued*

Deaths and Death Rates per 100,000 of White and Colored Population from Cancer in Registration States of 1920 for 1920-1923 Inclusive

AGE	SKIN				UNSPECIFIED			
	No. W.	No. C.	R. W.	R. C.	No. W.	No. C.	R. W.	R. C.
35-39	75	4	.5894	.3357	910	58	7.151	48.667
	47	7	.4079	.6219	657	56	5.701	4.975
40-44	129	9	1.244	1.0131	1,343	91	12.949	10.243
	85	10	.87171	.1207	1,189	92	12.193	11.045
45-49	204	7	2.0872	.7273	2,086	136	21.343	14.130
	89	7	1.0353	1.0525	1,506	97	17.520	14.585
50-54	330	16	4.0811	2.4279	3,150	161	38.956	24.432
	128	14	1.7513	2.8716	2,029	107	27.761	21.949
55-59	395	16	6.3477	4.1511	3,946	143	63.413	37.098
	201	16	3.5188	5.5476	2,139	88	37.446	30.512
60-64	614	10	11.785	3.0747	5,006	176	96.077	54.117
	282	10	5.9303	4.0327	2,370	81	49.838	32.664
65-69	765	21	21.863	9.5586	5,090	129	145.45	58.719
	337	12	10.168	6.8786	2,210	70	66.685	40.127
70-74	894	4	38.729	2.877	4,894	117	212.02	84.168
	497	14	21.411	10.968	1,831	60	78.879	47.013
75-79	968	8	69.227	9.996	3,483	77	249.09	96.246
	540	7	36.065	9.410	1,356	31	90.567	41.668
80-84	699	3	114.22	8.101	1,733	40	283.16	108.01
	551	6	74.644	14.14	781	22	105.81	51.835
85	449	1	199.48	6.327	696	19	309.19	120.17
	448	2	151.85	10.53	328	10	111.18	52.624

The statistics on cancer of the breast show that whites are more apt to be affected than the negroes. It is hardly probable that this is due to more frequent failures to recognize cancer of the breast in the colored population. There may, however, be causes other than race which might account for the difference, because among colored women the mammary glands probably function more nearly in a normal manner than among white women. It is a curious fact that spinsters are more apt to suffer from cancer of the breast, while they are much less apt to have cancer of the uterus than women who have had children. There seems to be something connected with the nonfunctioning of the mammary glands which disposes them to become cancerous. The racial differences in the incidence of breast cancer, however, are consider-

ably greater in the advanced age groups when the functioning of the mammary glands is past, and it is least in the period of active lactation. The situation is the reverse of what occurs in relation to cancers of the female generative organs, which in the period of child bearing are about twice as prevalent in colored females as in white females, whereas after age seventy they are only about 25 per cent more common. It seems not improbable that the low rate for cancer of the breast among colored women is due to the greater resistance of the negro ectoderm to various infections and injuries, but the evidence on this point is not conclusive. In the male cancers of the breast are too rare to furnish any statistically significant comparisons.

The most striking difference in the cancer mortality of the two races is found for cancer of the skin. In this case the difference cannot be explained as a matter of diagnosis. The number of deaths due to cancer of the skin is so small as to render the rates for the several age groups unreliable, nevertheless a glance at the table will show a marked tendency for the rates of the whites to be higher at all periods and especially in the advanced ages. The data from the Metropolitan Life Insurance Company and those derived from the U. S. Army during the great war corroborate this conclusion. It seems to be quite evident that the rarity of skin cancer in the negro is a true racial trait, like his relative freedom from cutaneous diseases in general.

Whether the relatively low mortality of the negro from cancer of the buccal cavity is due to racial heredity is more uncertain. Among males the whites show a greater rate for buccal cancer in nearly all age groups, but in the females the racial differences are slight. The total number of cases is small, but the data are sufficient to show two things: the higher rate in white males and the much lower rate for females in both races. Inasmuch as a good deal of the cancer of the buccal cavity is probably due to smoking, which is doubtless even now more prevalent in the male sex, it may be possible to explain the greater frequency of buccal cancer in white males as the result of habits rather than race.

RICKETS

Deaths diagnosed as caused by rickets are relatively few in number. Nevertheless this not uncommon disease is probably responsible indirectly for a considerable number of deaths in infancy and childhood. Almost all the deaths occur under five years of age, and considerably over half of them occur during the first year. Adding the deaths in the

Registration States for 1920 to 1923 gives 1,408 deaths among the whites and 500 among the colored. Taking those falling within the first year we find 924 whites and 284 colored. The death rates per 100,000 population are .428 for the whites and 1.59 for the colored, a rate over three times as high for the latter group.

Whether or not there is a racial diathesis to rickets cannot be decided. Syphilis, tuberculosis, and unfavorable conditions of life may, so far as we know, entirely account for the relatively high death rate from this disease in the colored population. Dr. G. N. Acker ('94), who has paid especial attention to the early symptoms of rickets which ordinarily escape record, makes the rather broad statement that "Negroes are almost without exception rachitic. This predisposition to rickets in the colored race would seem to be an acquired one, for it is stated that the native African seldom, if ever, shows any evidence of this disease." F. T. Mitchell [1] who has examined 500 colored and 500 white children from Memphis, Tenn., finds evidence of rickets in 87.6 per cent of the colored and in 49.8 per cent of the white. Marked signs of rickets occurred in 11.2 per cent of the white children and 34.6 per cent of the colored.

NEPHRITIS, ACUTE AND CHRONIC

The death rate from nephritis is uniformly higher among the colored population. This statement is true for deaths under one year as it is for the various higher age groups. The death rates of the white and colored population for the Registration States of 1920 are shown in Table 19.

The experience of the Metropolitan Life Insurance Company parallels these statistics from the Registration States except that the death rates for insured workers are higher than those for the general population. The excess of the death rate in the colored workers is found for "each of the sexes and for every age." The mortality of males is considerably greater than that of females. We might be prone to attribute this to alcohol were it not for the fact that the male excess is pronounced in the first year of life and during childhood as well as in old age. The excess of negro mortality is especially noticeable in the first year of life. During the period from 1914 to 1923 there were in the Registration Area 4,646 deaths among white infants and 683 among the colored, giving a ratio of 6.80 whites to 1 colored as compared with

[1] *So. Med. Jour.*, 23, 228-237, 1930.

TABLE 19

Death Rates from Nephritis per 100,000 Population White and Colored in the Registration Area 1920 (1920-1927)

	1920		1921		1922		1923		1924		1925		1926		1927	
	W	C	W	C	W	C	W	C	W	C	W	C	W	C	W	C
All Ages—Adjusted	78.8	131.1	74.6	130.9	74.4	137.3	78.8	140.9	76.4	158.0	84.9	179.0	88.5	147.1	82.2	174.2
All Ages—Crude	86.7	111.5	82.5	111.7	86.3	116.7	88.3	120.6	86.2	137.1	93.3	150.1	97.3	130.3	90.5	146.1
Under 1 Year	29.9	50.5	25.0	47.0	24.8	51.2	18.7	39.0	21.7	44.9	18.5	55.8	15.9	25.8	14.4	28.3
1 to 4	8.5	14.8	8.1	18.6	6.8	19.7	6.7	19.2	6.3	16.1	5.8	20.5	5.4	10.9	4.7	13.6
25 to 34	18.4	45.4	17.0	52.9	16.1	41.4	17.2	52.5	16.4	52.3	15.8	58.7	15.6	53.7	15.3	60.1
65 to 74	643.9	933.8	609.7	886.9	646.0	940.5	668.7	967.0	644.5	1075.1	734.2	1212.1	783.5	940.5	723.9	1126.6

an expected ratio of 7.47 (white to colored) based on deaths from all causes, thus showing that negro deaths from nephritis exceeded the expected number in the first year of life.

DIABETES MELLITUS

In contrast to nephritis, diabetes takes a markedly higher toll from the whites than from the colored population. During the years 1920 to 1927 the rates (per 100,000) for the Registration States for 1920 were as follows:

		1920	1921	1922	1923	1924	1925	1926	1927
White	Crude	16.9	17.6	19.7	19.0	17.4	17.9	19.3	18.7
	Adjusted	15.9	16.5	18.3	17.5	16.0	16.8	18.1	17.6
Colored	Crude	8.0	8.3	9.5	10.2	11.2	11.5	11.0	13.4
	Adjusted	9.6	9.8	11.3	12.1	13.2	13.9	12.7	16.3

The excess of deaths among whites occurs in all the age groups from infancy to old age, but it tends to become relatively greater in the advanced periods of life. This is shown also by the statistics of insured wage earners of the Metropolitan Life Insurance Company for 1911 to 1916. In both races and in both sexes there is a relatively sudden increase in the death rate from diabetes between age groups 35 to 44, and 45 to 54 years, after which the rate rapidly rises to old age. In the earlier years of life male deaths tend to preponderate over female deaths, but after 45 years the rate for females suddenly exceeds that of the males and continues relatively high through the remaining years of life in both the white and colored population. This same tendency is shown for both races in the differently situated wage earning population insured by the Metropolitan Life Insurance Company.

As is well known, food habits have much to do with the prevalence of this disease, and they may explain most of the differences in the mortality of the two races from this cause. The general death rate from diabetes has been on the increase during the last three decades, and even the use of insulin has produced no marked diminution in the mortality from this disease. In the recent experience in the Metropolitan Life Insurance Company "the death rate from diabetes among insured negroes . . . . has actually exceeded that of the whites."

DISEASES OF THE DIGESTIVE SYSTEM

For diseases of the digestive system the colored population compares quite favorably with the whites. The colored population does not fill its quota of deaths from appendicitis, hernia, diseases of the liver,

ulcer of the stomach and duodenum, and diseases of the pancreas. On the other hand, it has exceeded its quota for diseases of the stomach (cancer and ulcer excepted), peritonitis (without specified cause),—owing perhaps to faulty diagnosis,—and diseases due to intestinal parasites other than hookworm. By far the largest single category of deaths is "diarrhea and enteritis." Most of these deaths occur in the first year of life and they are set apart as a separate category in our mortality statistics. The number of young children dying of these complaints fluctuates exceedingly according to seasons and geographical environment. Maryland during the past few years has shown the highest rate for these diseases and about the highest excess of deaths among the colored population.

The reduction in the death rate from diarrhea and enteritis constitutes an important factor in the general decline of infant mortality which has occurred in the last two decades. Relatively the death rate in cities has fallen in both races more rapidly than it has in the rural districts. Death rates from diarrhea and enteritis are higher among the negroes than among the whites for all age periods. The statistics of the Metropolitan Life Insurance Company for insured laborers (largely an urban population) agree substantially with those from the Registration States.

The negro death rate from "dysentery" in the Registration Area is relatively much higher than that for whites, but this is mainly due to the circumstance that a large proportion of such deaths are reported from the South where the rate for both races is much higher than in the North. Dysentery is a very unsatisfactory category of diagnosis since it includes infections of both the amoebic and bacillary type, and many cases more properly attributable to diarrhea and enteritis, tuberculosis, and various other diseases.

DISEASES OF THE HEART

In recent mortality statistics diseases of the heart are responsible for by far the largest number of deaths attributed to any single cause. Formerly they were outnumbered by deaths from tuberculosis, but since 1912 deaths from heart disease have become the more numerous, and owing to the fact that the death rate from heart disease has been increasing while the death rate from tuberculosis has been decreasing the present disparity has become very marked.

A part of the statistical increase in deaths from heart disease as shown by the figures from the Registration Area is spurious. Since

the population is coming to be composed of a larger proportion of persons in the higher age groups, in which heart disease is more prevalent, the crude death rate would naturally tend to rise. Recently physicians have become much better equipped to diagnose heart disease than they were twenty years ago. Changes in the classification of causes of death have also caused a number of maladies to be assigned to heart disease that were previously not so classed. Dropsy, for instance, has largely disappeared as a reported cause of death, and the cases have gone in part to swell the mortality attributed to diseases of the heart. A glance over Howard's instructive table of deaths from cardiac and renal diseases in Baltimore shows a gradual increase of the rates from heart disease from the absurdly low figures in the first half of the nineteenth century (it was not recognized at all before 1824) to 1918 when it reached its highest point.

Another factor in the statistical increase in mortality from heart disease is due to the fact that the Registration Area as it has enlarged has come to include more and more states having a large negro population, among which heart disease is much more prevalent than it is among the whites. The marked excess of negro mortality is shown in all states and cities in which adequate records have been compiled. In general it averages roughly about 50 per cent higher than that of the whites. It is markedly higher in cities than in rural districts for both the colored and white population, but the excess of heart disease in the colored is greater relatively than it is in the whites. In other words, urban life tends to affect the colored population more unfavorably than the whites so far as affections of the heart are concerned.

Lower rates for the whites are shown in all age periods up to the advanced age groups (75 and over) when the scale turns in favor of the colored population. Undoubtedly syphilitic infection is an important factor in the high negro death rate from cardiac affections. Dublin in reviewing a large series of data states that "It is especially among the colored patients that syphilitic heart disease is prominent; in this group it accounts for about a third of the cases." Whether other factors favor the development of heart and circulatory disorders more on the whole in the negroes than in the whites is uncertain.

The excess of heart disease among the negroes is all the more significant because the age composition of the two races would favor a much higher mortality from this cause among the whites. When adjusted instead of crude rates are compared it is found that adjusted rates are higher than crude rates among the negroes, and that the differ-

ences in the mortality of the two races are greater for adjusted than for crude rates.

CEREBRAL HEMORRHAGE AND APOPLEXY

"Cerebral hemorrhage and apoplexy" constitute a cause of death which is closely related to diseases of the heart and like the latter it has come to occasion an increasing number of deaths in recent years. The age distribution of deaths is similar to that of organic diseases of the heart except that there are fewer deaths in childhood and adolescence. Cerebral hemorrhage and apoplexy are especially fatal to the urban negro. The death rate from these causes in the Registration Area is somewhat higher among the whites, but in the cities it is markedly higher among the colored population while in the rural districts the rate for the colored population is considerably below that of the whites. The death rates for the two years for which they have been calculated are as follows:

TABLE 20

Death Rates from Cerebral Hemorrhage and Apoplexy per 100,000 Population, White and Colored, in the Registration States, 1921, 1922

	1921		1922	
	White	Colored	White	Colored
Registration States	84.7	72.3	87.4	75.2
Cities in Registration States	81.3	98.5	83.5	113.1
Rural Districts	88.1	58.7	91.3	57.3
Registration Cities in Non-Registration States	66.1	115.8	60.3	94.3

According to the experience of the Metropolitan Life Insurance Company negroes have a higher mortality from cerebral hemorrhage and apoplexy than the whites. This obtains for all age groups except age 65 and over for men and age 75 and over for women. The higher rate among the negro industrial policy holders of this company as contrasted with what is found in the general population of the Registration Area may be due to predominantly urban life of the insured group.

DISEASES OF THE SKIN

As a rule diseases of the skin are less fatal to negroes than to whites. The death rate from diseases of the skin and cellular tissues in the Registration States for 1920 (1920-24 inclusive) was 4.30 per 100,000 for the colored and 3.63 for the white population, but the somewhat higher rate for the colored population is due largely to the inclusion of

deaths from gangrene, which is not properly a skin disease. The colored population contributes a little more than its expected quota of deaths from gangrene, but much less than its expected quota of deaths from furunculosis. This is shown in the experience of the Metropolitan Life Insurance Company as well as in the mortality statistics of the Registration Area. Since fatal cases of furunculosis are often caused by a streptococcus invasion, the immunity of the negro may be partially due to the same factors that reduce his death rate from erysipelas and scarlet fever. "Acute abscess" and "other diseases of the skin and cellular tissues" show racial differences in mortality which are much less marked.

Facts of interest on the distribution of diseases of the skin in the two races were brought out during the examination of recruits for the late war. Love and Davenport state that "acute abscesses and inflammation of the connective tissue of the skin are about one-third as common in colored as white soldiers; boils are one-fourth as common; dermatitis from traumatisms are one-third as common, and venomous bites and stings have much less effect. As is well known, the reaction of the skin to cuts differs in the two races. Colored persons tend to form keloid tumors, or skin ridges along the scar." For the period of the war the admission rates for the more common skin diseases in the white and colored troops were as follows:

TABLE 21

Admission Rates for Skin Diseases per 1,000 White and Colored Troops in the U. S. Army, April 1, 1917-December 31, 1919

	WHITE	COLORED
Carbuncle	.59	.13
Furuncle	5.44	1.59
Abscess	4.17	3.15
Cellulitis	3.46	1.62
Defects of Nails	2.21	.64
Eczema	1.06	.50
Herpes	.80	1.03
Scabies	8.29	2.37
Psoriasis	.40	.09
Impetigo	.71	.21
Other Diseases	5.03	4.17

Deaths from skin diseases were too few in number to give significant rates.

DISEASES OF THE NERVOUS SYSTEM AND SENSE ORGANS

I have calculated from the data in the U. S. Mortality Statistics the death rates of the white and colored population in the Registration

States for 1920 for the five years from 1920 to 1924 inclusive. They are as follows

	1920	1921	1922	1923	1924
Whites	124.8	120.8	125.3	129.7	129.3
Colored	124.8	124.4	130.5	138.8	149.7

The data for 1920 are not strictly comparable with those of later years since they include a number of diseases which under the revised classification adopted in 1921 are no longer included under nervous diseases. Among these are encephalitis, meningitis, and poliomyelitis. While the death rate from nervous diseases for the past five years is higher among the negroes the ratio of deaths of whites to those of the colored is somewhat higher than the ratio of deaths from all causes, the ratio being 10.74 W to 1 C, while the expected ratio based on deaths from all causes is 7.78. The death rates from diseases of the nervous system in the Registration Area in 1890 and 1900 were as follows:

	1890	1900
White	243.0	213.7
Colored	332.9	294.6

A comparison of these rates with those for recent years shows that deaths from nervous diseases have undergone a remarkable decrease in both races during the past three decades. To a large extent this decline is due to changes in classification of causes of death.

Diseases of the nervous system show a somewhat higher rate for negro infants than for those of the whites. This is due mainly to the fact that convulsions have been included under nervous diseases and the fact that convulsions are much more prevalent among negro than among white infants. Obviously "convulsions" is a very unsatisfactory category under which to classify real causes of death. The true cause of convulsions may not lie in the nervous system at all since a number of disorders of the most diverse origin may terminate in convulsive seizures. More accurate specifications of the causes of death based on the primary source rather than the terminal symptoms of the reported death would materially reduce this category of diagnosis. Doubtless a part of the high negro mortality from convulsions is to be attributed to less precision in recording the cause of negro deaths. Another factor is the prevalence of rickets, which predisposes infants to convulsive seizures. The relatively high preponderance of negro deaths from epilepsy may be caused to a considerable extent by the greater prevalence of syphilis.

The data on insanity show,—if we can believe them,—that there has been an increase of the insane among the colored population since the period of slavery. I do not quote the figures because they are obviously unreliable. Hoffman has discussed data on mental disorders before and following the Civil War and has shown that these maladies were much more common among negroes after the war than they were under slavery. Considering the increase of alcoholism, tuberculosis, syphilis, and the general physical deterioration of the negro physique that followed emancipation this increase of insanity was doubtless inevitable.

The negroes, however, are not represented in asylums in numbers comparable to their proportion to the general population. According to the statistics compiled by H. H. Laughlin the negro fills little more than half his quota of the asylum inmates of the United States. The Census Bulletin 119 on *The Insane and Feeble Minded in Institutions (1910)* shows that negroes are relatively less represented in asylums than the whites. These facts, however, signify very little as to the relative prevalence of insanity in the two races.

It is often stated that there is less neurasthenia among negroes than among whites, and there were fewer cases among negroes who were recruited during the late war. There are also many statements to the effect that neural manifestations of syphilis are relatively uncommon among the negroes, and this is borne out by the data in our mortality statistics. On the other hand the Army data show a higher percentage of tabes and general paresis in the negro recruits. R. H. Foster ('26), who has endeavored to collect the statistics from all the hospitals which contain any considerable number of negroes, finds a markedly higher percentage of paresis among both male and female negroes, than among the whites. A number of recent writers have emphasized the relative frequency of tabes and general paresis in the negroes,—which is not surprising when we consider the large amount of syphilis in the negro race.

Negroes apparently suffer less than the whites from diseases of the ear. Otitis media, otitis externa, mastoiditis and defective hearing were much less common in negro than in white recruits during the great war. Dr. Scheppergrell ('95) on the basis of data covering over 40,000 cases states that "In chronic suppurative and non-suppurative inflammations of the middle ear, the ratio of the negro is also small, being 16 and 26 respectively to 100 cases in the whites." Possibly the negro's advantages in these respects may be due in part to his relative

freedom from scarlet fever, diphtheria, measles and affections of the nose and throat, which sometimes lead to infections of the ears as a secondary complication. They may also be in part due to certain peculiarities in the structure of the negro ear, as pointed out by Murrell ('87).

The immunity of the negro to trachoma has been both affirmed and denied.

PUERPERAL CAUSES

The death rate of the negroes from causes connected with childbirth averages higher than that of the whites. Even the death rate of foreign born white mothers of every nationality is exceeded by the rate for the colored population. Deaths from puerperal causes are uniformly much higher for both colored and white persons in the cities than in rural districts.

TABLE 22

Deaths from Puerperal Causes per 100,000 Live Births in Cities and Rural Districts of the Registration Area

		1918	1919	1920	1921	1922	1923	1924	1925	1926	1927
Cities in R. A.	W	9.3	7.6	8.2	7.4	7.0	7.1	7.0	7.0	7.1	7.0
	C	16.7	13.9	15.1	13.1	12.4	12.3	12.9	12.4	12.1	13.4
Rural part of R. A.	W	8.4	6.3	6.9	5.4	5.5	5.3	5.1	5.0	5.2	4.9
	C	12.5	11.8	11.7	9.7	9.8	10.2	11.1	11.1	9.7	10.1

One large factor in such deaths is puerperal septicæmia, which still shows an inexcusably high frequency. After the fall of the death rate from this disease following the discovery of its cause and the employment of precautions against infecting mothers during delivery, this scourge has lingered on with relatively little abatement for several years. For females during the child bearing period it still ranks, next to tuberculosis, among the chief causes of death. The rate for the colored population is nearly double that for the white as is shown in the table.

TABLE 23

Deaths from Puerperal Septicaemia per 100,000 Population, White and Colored, Registration States of 1920, 1920-1927

		1920	1921	1922	1923	1924	1925	1926	1927
White	Crude	12.4	12.8	10.5	11.5	10.5	9.9	9.7	9.4
	Adjusted	12.4	12.9	10.5	11.8	10.6	9.9	9.7	9.4
Colored	Crude	23.0	23.3	20.7	20.9	22.5	22.5	17.8	21.9
	Adjusted	21.6	21.8	19.5	19.6	21.0	21.1	16.9	20.6

The period of greatest mortality among the whites is 25 to 29 and among the colored 20 to 24.

Among other puerperal causes of death, which as a group take an even higher toll than puerperal septicæmia, the chief factor is puerperal albuminuria and this likewise is more common in the colored population. For most other fatalities associated with pregnancy the death rate among the colored population is also relatively high. There are several causes which conspire to bring about this result. Illegitimacy, syphilis, lack of adequate prenatal care, and of asepsis during delivery naturally enhance the negro death rate from most puerperal causes. If we can accept the statements in regard to the relative ease of child bearing on the part of negro mothers, whatever racial factors may be involved in the death rate from puerperal causes would probably be in favor of the negro. The relatively high death rate of colored females is doubtless to be attributed mainly to environmental causes.

MALFORMATIONS

In the light of Dr. Mall's studies upon the causes of malformations one would naturally suppose that the prevalence of tuberculosis and syphilis in the colored population would give rise to a relatively high proportion of anomalies leading to fatal results. I am inclined to set little store by the statistical data in this connection. It is probable that malformations would be designated as a cause of death much less frequently in the case of negro infants. Enumeration of the deaths in the Registration States for 1920 during 1920 to 1923 shows that the negroes fall far short of furnishing their quota, the ratio of deaths among white and colored being 19.48 to 1 instead of 7.78 to 1. The ratio of deaths under five, when most deaths occur, is 19.60 to 1. Malformations of the heart show a slightly higher ratio for the whites, namely 21.10 to 1 for all ages, and 20.0 to 1 for ages under five. The tabulation of a larger series of deaths under one year in the Registration Area, 1914 to 1923, gives 109,047 deaths among white infants and 4,675 among the colored, or a ratio of 23.41 white to 1 colored. The expected ratio based on all deaths is 7.47 to 1. In the same years and area the ratio for congenital malformations of the heart, based on 66,806 deaths among the whites and 2,759 among the colored, is 24.21 white to 1 colored. The ratio for hydrocephalus based on 7,722 white and 478 colored is 16.15 to 1, and for other congenital malformations based on 34,519 white and 1,428 colored is 24.17 to 1.

CONGENITAL DEBILITY, ATROPHY AND MARASMUS

The death rate from maladies grouped under this heading is relatively high for the colored population. Doubtless numerous deaths for which syphilis is primarily responsible are classed under this rubric. Infants predisposed to rickets, as so many are among the negroes, often die of disorders of this class. Deaths from prematurity are more common in colored than in white infants, but the ratio of deaths from this cause is less than the ratio from all causes combined.

Injuries at birth are relatively rare among negroes notwithstanding the relative infrequency of skilled obstetricians in attendance. Theoretically one might attribute this to the absence of obstetrical meddling with the normal process of delivery, but I am inclined to take a more generous view and to ascribe the result, at least in part, to the greater ease with which the negro mother gives birth to children. Doctor DeSaussure, who has made numerous observations on the delivery of negro infants, says that "labor in the negro is a simple act," and usually occasions little disturbance in the mother. The ease with which child bearing occurs among primitive races has frequently been referred to, but the matter has probably been often exaggerated. In commenting on this matter, Prof. J. W. Williams remarks (*Obstetrics*, p. 169-170) "It is generally believed that the comparatively difficult labors of the women of the upper classes are due to the enervating influences of civilization and luxury, while the easy labors of Negroes are considered as manifestations of a closer approach to Nature. Such conclusions are not justified by my experience, as the physical degeneration of colored women living in large cities is proven by the fact that in them the incidence of contracted pelvis is four or five times greater than in white women. Were this not counterbalanced by the lesser weight of the black children, and particularly by the smaller size and greater compressibility of their heads, labor would be a disastrous function, and would comparatively soon lead to a solution of the race problem. If the Negro children were as large and had as hard heads as in the upper classes, I should be obliged to perform cesarean section several times more frequently than I actually do."

STILLBIRTHS AND ABORTIONS

The high percentage of stillbirths and abortions among negroes is well known. Not only is this fact attested by several writers who have treated the subject specifically, but it is indicated by the rather extensive data on stillbirths and abortions published by the U. S. Census. Data

have now been published for the U. S. Registration Area for Births during eight years, 1918, 1922-1928, and they include a total of 481,451 cases among whites and 95,806 among the negroes. The ratios of abortions and stillbirths per 1,000 births were about twice as high for the negroes as for the whites.

Among urban negroes especially, the practice of abortion appears to be increasingly common. In commenting on the negroes of South Carolina, DeSaussure ('95) remarks that "abortions criminal and non-criminal are very common," and in agreement with many other writers he attributes a large proportion of stillbirths among negroes to syphilis. This disease he estimates is found in "70 per cent of the negroes of Charleston" Dr. G. H. Day estimates the proportion of syphilis as high as 75 per cent. Although these estimates are probably much too high, there is little doubt that syphilis is sufficiently prevalent among negroes to account for the whole excess of their rate for stillbirths and abortions.

EXTERNAL CAUSES

The death rate of the colored population from external causes is relatively high. Our colored people, however, show little proclivity to commit suicide, and their fatalities from automobile accidents (a not inconsiderable cause of death) are not as numerous relatively as those of the better circumstanced whites. The rate from miscellaneous sources of accidental deaths is higher for the colored population, and the rates for violent deaths are particularly high as may be seen in the following table of deaths for 1924:

TABLE 24

Death Rates from Violence in 1924 per 100,000 Population, White and Colored

	WHITE	COLORED
All Ages—Crude	80.8	132.6
All Ages—Adjusted	76.5	133.1
Under 1	104.0	256.4
1-4	73.5	135.9
Under 5	79.2	160.0
10-14	37.3	49.8
15-19	60.7	109.7
20-24	69.9	171.7
25-34	62.8	175.8
35-44	76.8	145.8
45-54	85.9	126.0

The most outstanding cause of deaths in this class is homicide. For several years the homicide rate in the Registration Area has been between five and eight times as high for the colored as for the white population. In 1924 it was 5.3 per hundred thousand for the whites and 40.1 for the colored, and if the statistics included only males the rate for the colored population would be relatively still higher. Speaking of the wage earners insured in the Metropolitan Life Insurance Company, Dr. Dublin states that "from 15-35 years of age, negro males have a rate approximately ten times that of white males. At these ages homicide ranks third as a cause of death, being exceeded only by the figures for tuberculosis and the acute respiratory diseases." The experience of this company from 1911 to 1916 gave the following homicide rates per 100,000 persons: Male white, 5.4; male colored, 52.2; female white, 1.9; female colored, 14.1.

For the past decade the homicide rate has shown no improvement. Hoffman in his discussion on *The Homicide Problem* has shown that in a group of 28 cities the homicide rate increased from 5.1 per 100,000 in 1900 to 10.3, the highest rate, in 1924. If these high rates continue and preserve their present distribution as to race, homicide will prove to be a not unimportant factor in the relative increase of the two races.

ILL-DEFINED CAUSES

The number of negro deaths attributed to "ill-defined causes" is very high. In 1921 the death rate was 86.9 per hundred thousand for negroes and 9.5 for whites. Of these deaths the largest category (over two-thirds of the total) consists of deaths from "non-specified or unknown causes." This means simply that there has been much more carelessness or inaccuracy in reporting causes of deaths in the colored population. A high proportion of unclassified deaths occur in infancy. These amount to nearly 10 per cent of the deaths among colored infants, and were these properly distributed it might make considerable difference in the statistics of the relative mortality of the two races from certain diseases.

INFANT MORTALITY

Infant mortality presents certain distinctive features which justify its consideration under a separate heading. It is influenced profoundly by environmental factors, as is evidenced by its striking decrease during the past three decades. The commonly employed measure of infant mortality,—the number of deaths under one year of age per thousand

born during the year,—depends for its accuracy not only upon complete registration of deaths, but also upon complete registration of births. The data on births collected during the censuses of 1880 and 1890 are so obviously incomplete as to be practically worthless, and the records of infant deaths fall very far short of the true number. Statistics on infant mortality covering a large part of the United States are of little value previous to 1900. The few states and cities which have kept reliable birth and death statistics for a period of several years before this date are mainly in the north, where the negro population was scanty. From various fragmentary sources, however, it is evident that infant mortality in the negro population has always been high, and it continues to be much above that of the whites, even where there has been the greatest decline in the death rate of negro infants. Even in 1890 the death rate of negro infants per thousand population was 430.2 in New Orleans; 461.7 in Charleston, S. C., and 529.8 in Richmond, Va. These appalling death rates were probably characteristic of the rank and file of cities having a large negro population. The negro birth rate in the south has been notoriously high, but notwithstanding this fact the high infant mortality cut down the rate of natural increase so that the proportion of children under five years of age among the whites has been exceeding that among the negroes to an ever increasing extent.

For the census years 1900 and 1910 infant mortality rates have been estimated for the two races on the basis of the number of infant deaths in the U. S. per thousand population under one year of age. These are as follows:

	1900	1910
White	159.4	129.7
Negro	344.5	261.9

The U. S. Mortality Statistics now furnish us with full data on the number of deaths of white and colored infants for various diseases in the Registration Area for Deaths. Since 1916 the reports on birth statistics have published records of the death rates of white and negro infants per thousand births in the expanding Registration Area for Births, and have given full data on infant mortality from various diseases for several sub-divisions of the first year of life. Adequate means for studying the course of infant mortality in our white and colored population have therefore been available for only a few years.

TABLE 25

Deaths and Ratios of Deaths of White and Colored Infants in the Registration Area, 1911-1923

CAUSES OF DEATH	NO. OF DEATHS W	NO. OF DEATHS C	RATIO OF W TO C
All causes	1,447,538	193,658	7.474
I. General diseases	158,206	31,565	5.012
1. Typhoid fever	431	182	2.368
5. Malaria	1,265	1,378	.918
7. Measles	14,276	1,305	10.939
8. Scarlet fever	1,500	54	27.77
9. Whooping cough	36,394	6,696	5.435
10. Diphtheria and croup	8,047	1,128	7.133
11 a and b. Influenza	35,375	6,817	5.189
16. Dysentery	5,381	2,433	2.2117
21. Erysipelas	7,498	248	30.23
22. Poliomyelitis	2,008	168	11.95
24. Meningococcus Meningitis	3,078	214	14.38
29. Tetanus	1,798	1,575	11.41
31-37. Tuberculosis	18,743	3,129	5.990
31. Respiratory tuberculosis	5,507	1,715	3.211
32. Tuberculous meningitis	8,981	863	10.40
33. Abdominal tuberculosis	1,508	147	10.25
37 a and b. Disseminated tuberculosis	617	271	2.276
38. Syphilis	14,598	4,967	2.939
40. Gonococcus infection	486	81	6.0
41. Purulent infection and septicæmia	1,525	91	16.75
43-49. Cancer and malignant tumor	520	.....	....
III. Diseases of the Nervous System	35,064	5,207	6.734
71a. Simple meningitis	7,983	569	14.03
74a. Cerebral hemorrhage	1,330	147	9.048
78. Epilepsy	817	258	3.166
80. Convulsions	1,613	3,448	.468
86. Diseases of the ears	2,940	171	17.19
IV. Diseases of the Circulatory System	11,046	675	16.36
88. Acute endocarditis	2,949	125	23.59
90. Other diseases of the heart	5,293	368	14.38
94. Diseases of lymphatics	1,771	110	16.10
V. Diseases of respiratory system	221,889	33,032	6.717
98. Diseases of the larynx	1,375	199	6.91
99 a, b, c. Bronchitis	27,587	3,341	8.257
100 a, b. Broncho-pneumonia (capillary bronchitis since 1921)	129,342	16,348	7.911
101 a, b. Pneumonia, lobar and undefined	56,194	11,251	5.058
102. Pleurisy	1,634	123	13.28

TABLE 25—*Continued*

Deaths and Ratios of Deaths of White and Colored Infants in the Registration Area, 1911-1923

CAUSES OF DEATH	NO. OF DEATHS W	C	RATIO OF W TO C
VI. Diseases of digestive system	320,993	34,670	9.258
113. Diarrhea and enteritis	284,381	29,635	9.596
118a. Hernia	1,440	261	5.52
118b. Intestinal obstruction	8,832	745	11.85
VII. Non-venereal diseases of the genito-urinary system:			
128-129. Nephritis, acute and chronic	4,646	683	6.803
IX. Diseases of the skin	5,844	275	21.25
XI. Malformations	109,047	4,675	23.32
159a. Hydrocephalus	7,722	478	16.15
159b. Congenital malformations of the heart	66,806	2,759	24.21
159c. Other congenital malformations	34,519	1,428	24.17
XII. Early infancy	517,314	54,949	9.414
160. Congenital debility, atrophy, marasmus	89,685	13,793	6.502
161a. Premature birth	313,425	33,223	9.434
161b. Injuries at birth	67,603	3,309	20.43
XIV. External causes	17,106	3,299	5.185
197-199. Homicide	1,231	175	7.034
XV. Ill defined diseases	32,902	19,061	1.726
204. Sudden death	1,517	614	2.47
205b. Not specified, or unknown	25,371	20,067	1.264

I have given in Table 25 a summary of the data on infant mortality in white and colored infants in the Registration Area for Deaths from 1914 to 1923 inclusive, together with the ratios of deaths in white and colored infants. Under the head of "colored" are included infants of all colored races, but as only a small percentage of these are of Indian or Mongolian parentage, the data are fairly representative of the mortality of negroes. In the absence of data for the computation of infant mortality rates I have compared the ratios of deaths, (white to colored) for various causes as a means for comparing the relative mortality of the two races. The ratio of deaths (white to colored) from all causes during the entire period in question (1914 to 1923) is 7.474 to 1. This is a convenient number to keep in mind when comparing the relative mortality of the races from particular diseases, but it should not be used except in relation to the data for this area and

period. An inspection of the ratios in the right hand column of the table will reveal at once whether the death rate for a given disease is higher or lower than the expected rate based on all causes of deaths. A number higher than 7.47 indicates that the infant mortality rate for the whites is relatively higher for this disease.

TABLE 26

Mortality Rates for White and Negro Infants in the Birth Registration Area 1916-1928 (Deaths per 1,000 Living Births)

CAUSES OF DEATH		1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	1926	1927	1928
Measles	W	1.4	1.2	1.0	0.5	1.0	0.5	0.6	1.2	0.7	0.3	1.1	0.4	0.6
	N	.9	2.2	.8	0.2	0.4	0.4	0.5	1.8	1.4	0.2	1.0	0.4	1.1
Scarlet fever	W	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
	N	0.1	...	...	(1)	(1)	0.1	(1)	(1)	(1)	(1)	(1)	(1)	(1)
Whooping cough	W	2.2	2.2	3.1	1.2	2.9	2.0	1.3	2.1	1.8	1.6	2.1	1.7	1.4
	N	5.6	5.8	8.1	3.2	4.7	4.4	2.9	5.2	5.1	3.1	3.8	4.3	2.8
Diphtheria	W	0.5	0.5	0.4	0.4	0.4	0.4	0.5	0.4	0.3	0.3	0.2	0.2	0.3
	N	1.1	0.9	0.7	0.7	0.6	0.8	0.6	0.7	0.5	0.4	0.4	0.3	0.4
Dysentery	W	0.2	0.3	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.3	0.2	0.2	0.3
	N	0.4	1.8	1.9	2.0	1.9	1.2	1.3	1.1	1.1	0.9	0.8	0.8	1.0
Influenza	W	0.8	0.8	6.3	3.7	2.3	0.6	1.6	2.0	1.0	1.6	2.2	1.3	2.5
	N	1.3	1.0	13.0	10.6	6.9	1.2	2.9	4.7	2.4	3.3	4.7	2.6	4.6
Erysipelas	W	0.6	0.5	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
	N	0.5	0.2	0.2	0.1	0.2	0.2	0.1	0.1	0.2	0.2	0.2	0.1	0.1
Meningitis	W	0.8	0.8	0.8	0.7	0.6	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.2
	N	1.0	0.9	0.8	0.7	0.7	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Tetanus	W	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	(1)	(1)	0.1	0.1
	N	0.4	0.7	0.6	0.7	0.7	0.4	0.5	0.6	0.5	0.5	0.4	0.5	0.5
Respiratory Tuberculosis	W	0.5	0.5	0.4	0.4	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1
	N	2.3	1.4	1.6	1.2	1.0	0.8	0.7	0.7	0.7	0.6	0.6	0.7	0.6
Tuberculous Meningitis	W	0.8	0.7	0.6	0.6	0.5	0.4	0.4	0.4	0.3	0.3	0.3	0.2	0.2
	N	1.9	0.8	0.5	0.5	0.5	0.4	0.4	0.5	0.3	0.4	0.4	0.3	0.3
Other forms	W	0.2	0.2	0.2	0.2	0.1	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1
	N	0.3	0.3	0.2	0.1	0.2	0.2	0.3	0.3	0.2	0.2	0.2	0.2	0.3

TABLE 26—*Continued*

Mortality Rates for White and Negro Infants in the Birth Registration Area 1916-1928 (Deaths per 1,000 Living Births)

CAUSES OF DEATH		1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	1926	1927	19
Syphilis	W	1.2	1.0	0.8	0.8	0.7	0.7	0.7	0.6	0.7	0.5	0.5	0.5	
	N	7.9	3.9	3.5	2.8	2.8	2.8	2.7	3.0	2.8	3.0	2.9	3.2	
Convulsions	W	1.2	1.0	1.1	0.9	0.9	0.7	0.7	0.6	0.6	0.6	0.5	0.4	
	N	3.2	2.8	2.7	2.3	2.1	2.0	2.1	1.7	1.7	1.3	1.2	1.1	
Bronchitis	W	2.5	2.1	1.9	1.8	1.7	1.0	1.2	1.0	0.8	0.7	0.7	0.5	
	N	6.0	3.6	4.2	2.4	2.7	1.3	1.3	1.7	1.4	1.3	1.2	0.9	
Pneumonia	W	3.9	3.9	4.5	3.0	2.8	2.2	2.7	2.5	2.4	2.3	2.6	1.9	
	N	7.5	9.2	10.2	6.7	7.8	5.1	6.5	6.5	6.6	5.6	6.5	5.1	
Broncho-pneumonia	W	9.2	8.0	8.6	7.6	7.6	6.0	7.5	7.3	6.5	6.4	7.4	5.3	
	N	23.8	13.6	15.7	9.6	12.7	9.0	10.6	12.9	12.1	12.6	14.1	10.3	
Diarrhea and Enteritis	W	23.7	19.5	18.6	15.4	14.6	13.3	11.4	11.2	8.8	10.7	9.3	7.3	
	N	39.6	28.7	25.2	20.0	19.8	16.3	15.1	15.3	14.4	17.0	14.4	12.2	
Malformations	W	6.8	6.4	6.6	6.5	6.5	6.4	6.7	6.6	6.8	6.5	6.5	6.0	
	N	6.5	3.5	3.7	2.9	3.2	2.8	2.6	2.9	2.9	3.2	3.1	2.4	
Congenital debility	W	9.9	8.4	8.9	7.8	7.3	4.1	3.7	3.7	3.3	3.2	3.1	2.6	
	N	18.7	15.4	15.8	13.2	13.2	7.2	6.6	6.8	6.1	6.3	5.9	4.9	
Premature birth	W	19.1	19.0	20.1	18.6	18.9	17.6	17.8	17.5	17.2	16.9	17.4	16.6	
	N	31.6	21.7	21.3	26.7	26.3	22.3	22.1	22.2	23.6	21.3	21.3	19.2	
Injuries at birth	W	4.2	3.9	3.4	3.5	3.9	4.4	4.7	4.8	5.0	5.1	5.1	5.0	
	N	4.3	2.4	2.4	2.3	2.2	2.2	2.6	2.7	2.9	3.2	3.1	3.2	
Other diseases of infancy	W	...	...	...	...	...	2.7	2.5	2.5	2.4	2.3	2.1	1.9	
	N	...	...	...	...	...	3.2	3.2	3.3	3.2	3.4	3.1	2.9	
External causes	W	1.0	1.2	0.9	0.9	1.0	0.9	0.9	0.9	0.9	1.0	1.0	0.9	
	N	3.0	2.8	2.5	1.9	1.9	2.0	1.8	1.7	2.1	1.9	2.0	2.0	
Unknown	W	0.8	2.2	2.0	1.9	1.7	...	1.5	1.4	1.4	1.2	1.3	1.7	
	N	5.3	15.0	16.6	13.4	12.8	...	15.2	14.5	13.4	13.7	13.3	15.8	
Other causes	W	5.6	4.6	4.0	4.0	4.1	6.1	5.0	4.9	4.8	4.9	4.9	4.6	
	N	9.4	7.8	7.1	7.4	8.1	22.3	7.4	7.2	6.8	7.3	6.5	5.7	

(1) Less than one-tenth per 1 per 1,000 births.

Table 26 gives the death rates of whites and negroes from particular causes for individual years since 1916. In these data, which are compiled from the statistics on infant mortality published in the Birth Statistics, the deaths of negroes are kept separate from those of other colored people. The rates are based on the proportion of infants dying under 1 year of age to one thousand of the population. The table serves for making a rough comparison of the death rates of negro and white infants from different diseases.

It is apparent from both tables that the death rates of white and negro infants from different causes vary widely. The negro death rate is relatively low for measles, scarlet fever, erysipelas, malformations, diseases of the skin, and injuries at birth. It is relatively high for general diseases, typhoid, malaria, whooping cough, tuberculosis, syphilis, convulsions, lobar pneumonia, and external causes. The very high rate for "unspecified or unknown causes" as compared with that for white infants is simply an index of the greater proportion of inadequate diagnoses.

CONCLUSION

From what has been said it is evident that negroes react quite differently from the whites to several kinds of disease. Doubtless the differences would be still greater were it not for the extensive infusion of white blood into the negro race. Despite the numerous assertions which have been made as to the physical weakness of the mulattoes I can find no valid grounds for concluding that they are constitutionally inferior to either the whites or the pure blacks. So far as our meager evidence goes, mulattoes seem to be intermediate in their reactions to disease as they are in the color of their skin.

In dealing with statistics on negro mortality one is continually haunted by doubts as to the extent to which the data are reliable. Studies of the causes of deaths as revealed by autopsies as compared with the causes stated on death certificates have shown that physicians are not always infallible in their statements concerning why people die. That the data we have been compelled to employ contain many errors goes without saying, and they exhibit internal evidence of being less reliable for the mortality of negroes than for that of whites. Nevertheless the statistics on the mortality of the two races show a remarkable degree of consistency from year to year. To a certain extent this may be a consistency in error which future knowledge may furnish the means of correcting.

Upon casual inspection it would seem that the negro's immunities to disease are distributed without much rhyme or reason. The negro is peculiarly immune to diseases of the skin and to certain infections such as scarlet fever, diphtheria, measles, erysipelas, chicken pox and affections of the nose and sinuses, while he seems remarkably prone to contract infections of the respiratory tract. There is no adequate evidence that he has any less capacity to resist disease in general than the whites. The mortality of the negro is so greatly affected by his unfavorable environment and habits of life that for most diseases it is quite impossible to detect any influence of hereditary racial factors which may nevertheless be present.

In a previous paper ('28) I have made the suggestion that most of the negro's immunities to disease may be interpreted as due to the superior resistance of his ectoderm. The pigment of the skin affords a direct protection against the injurious effects of light, but it seems probable that in other respects pigment is more of an index of a physiological power of resistance than a means of defense *per se*. The greater thickness of the negro ectoderm not improbably makes it better able to resist invasion by micro-organisms. Since the nose, sinuses and a part of the mouth cavity are lined with ectoderm which has been invaginated during embryonic development, the negro may be somewhat better able to resist initial infection by those diseases which make their first inroads in the nasal or oral cavities. It is probable that the organisms which are responsible for scarlet fever, diphtheria, measles and common colds begin their active proliferation in the mucous membranes of the nose and throat. The germs of pneumonia, on the other hand, may lie in the oral cavity and do no damage until they gain access to the respiratory tract. Whooping cough, like the typical respiratory diseases, may also be primarily an entodermic infection. As to the first inroads of the primary offenders in influenza we are quite uncertain. It seems probable that the negro is less apt to contract this disease than the whites, but his greater mortality is due to his susceptibility to pneumonia. There are certain immunities of the negro which do not fall under the interpretation just given, such as his greater tolerance of hookworm infection, but most of them may be plausibly explained in accordance with this viewpoint.

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STUDIES ON HUMAN LONGEVITY

IV. THE INHERITANCE OF LONGEVITY

PRELIMINARY REPORT.[1,2]

BY RAYMOND PEARL

I

There has long and widely prevailed an idea that inheritance plays a part in human longevity. This idea undoubtedly arose from the common observation that length of days tends to run in families.[3] Oliver

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[1] From the Department of Biology of the School of Hygiene and Public Health, Johns Hopkins University.

This paper was read at a meeting of the Society of Hygiene, Johns Hopkins University, on March 18, 1931. It embodies, in its present form, some of the results of a coöperative study made by certain of the members of the writer's seminar in biology during the session of 1930-31. Those in the seminar taking part in this work, besides the writer, were Dr. John Rice Miner, Dr. I-chin Yuan, Mr. Takeo Imai, and Miss Ruth DeWitt Pearl.

[2] Earlier papers in this series of studies, which originally appeared without the general title, were:

I. Pearl, R. A note on the inheritance of duration of life in man. *Amer. Jour. Hyg.*, Vol. 2, pp. 229-233, 1922.

II. *Id.* Preliminary account of an investigation of factors influencing longevity. *Jour. Amer. Med. Assoc.*, Vol. 82, pp. 259-264, 1924.

III. *Id.* Longevity: A pedigree. HUMAN BIOLOGY, Vol. 3, pp. 133-137, 1931.

[3] Thus, in a discussion at the Anthropological Institute, R. S. Charnock said: "There is no doubt that longevity is hereditary. Rust, the physiologist, had never known an octogenarian in whose family there were not frequent cases of longevity." (*Jour. Anthrop. Inst.*, Vol. 2, p. 89, meeting of March 18, 1872). A more recent example is the following statement by David Riesman: "Heredity to a great extent determines longevity. Longevity as well as its opposite is largely an inherited trait. Not enough attention has been paid to this by geneticists, but it is as striking a familial trait as the color of the eyes, the conformation of the head, or any other physical feature." (*Jour. Amer. Med. Assoc.*, Vol. 96, p. 1109, 1931).

Wendell Holmes [4] expressed the matter neatly, in beginning a discourse of advice on the method of attaining the age of "three score years and twenty":

> The first thing to be done is, some years before birth, to advertise for a couple of parents both belonging to long-lived families. Especially let the mother come of a race in which octogenarians and nonagenarians are very common phenomena. There are practical difficulties in following out this suggestion, but possibly the forethought of your progenitors, or that concurrence of circumstances which we call accident, may have arranged this for you.

A concrete example of just such a family as that postulated has recently been given in this journal.[5]

Oddly enough the idea that inheritance plays an important rôle in longevity has been more taken for granted than supported by exact scientific investigation. Even a business so precise in some particulars as life insurance, which has, from its beginnings, acted on the assumption that the duration of life of an individual's near kinsfolk is of importance in estimating the nature of its risk accepted in insuring him, has made singularly little effort to determine exactly the weight of this factor.[6] One of the chief reasons why research on the problem of the inheritance of life duration in man has lagged as it has, is the difficulty of the problem, and particularly the difficulty of getting material of the kind and amount necessary for the effective discussion of the problem.

The two most important contributions to this field of research, those

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[4] Holmes, O. W. Over the Teacups (Standard Library Edit., Houghton Mifflin Co.), p. 181.

[5] Pearl, R. Longevity: a pedigree. HUMAN BIOLOGY, Vol. 3, pp. 133-137, 1931. For other examples see an earlier paper by the same writer: On the inheritance of duration of life. *Memorial Publ. 100th Birthday J. G. Mendel*, Prague, 1925, pp. 175-192.

[6] One probable reason why life insurance companies have not more extensively embarked upon this and various other biological investigations pertinent to their business, is not far to seek. The prime essential of the life insurance business is safety. This can be, and is, assured by the process technically known as "loading" the premium rates calculated from tables of mortality experience, to a degree sufficient to provide a margin of safety adequate to meet any eventuality. This can be done without any precise knowledge of the biological factors influencing each particular risk. Under these circumstances insurance companies have probably been wise in not embarking upon research programs with the policy-holders' money, similar in magnitude to the research efforts of other industrial concerns of comparable financial position.

of Beeton and Pearson,[7] and of Alexander Graham Bell,[8] were based upon data recorded in published genealogies. Indeed such material is practically the only sort that is available for the study of the problem, unless special data for the purpose are collected *de novo*. But any genealogical material so far used has serious defects for this particular purpose. Critical study of the earlier contributions referred to leads one to the position that while the conclusions reached by Pearson and by Bell regarding the inheritance of longevity may be true, and in their broader aspects, at least, probably are, the evidence which they present is quite inadequate to prove them. One serious difficulty about the genealogical material used by these writers is that, in both cases, the age distributions of the data, as wholes, are widely deviant from the age distribution of deaths in a normal population, so much so that the material cannot be reasonably regarded as a representative random sample from the general populations from which it was derived. In my first discussion[9] of this work this discrepancy was noted in the case of Bell's monograph, but less importance was there attached to this departure of the material from the usual condition than I should now regard as justifiable, after longer study of the problem. Unpublished studies of genealogical material which have been carried on in this department during the past ten years lead us to the view that the data recorded in any single genealogy (*i. e.*, for a single family group) are likely to show deviations from the general population in this actuarial characteristic. It would seem that the only possible hope of making any effective use of such material is by combining the data from a series of genealogies chosen at random,[10] and even then there may be found differences from a normal population.

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[7] Beeton, M., and K. Pearson. Data for the problem of evolution in man. II. A first study of longevity, and the selective death rate in man. *Proc. Roy. Soc.* (London), Vol. 65, pp. 290-305, 1899.

Id. On the inheritance of the duration of life, and on the intensity of natural selection in man. *Biometrika*, Vol. 1, pp. 50-89, 1901.

[8] Bell, A. G. The Duration of Life and Conditions associated with Longevity. A Study of the Hyde Genealogy. Washington (privately printed), 1918. Pp. 57. 4to.

[9] Pearl, R. The Biology of Death. Philadelphia and London (J. B. Lippincott Co.), 1922. Chapter VI *passim*.

[10] This difficulty regarding the use of genealogical material for such studies has been discussed by Professor Edwin Bidwell Wilson of Harvard University and C. R. Doering. Cf. The elder Peirce's. *Proc. Nat. Acad. Sci.*, Vol. 12, pp. 424-432, 1926.

Long thought and study of the problem of the inheritance of human longevity has engendered some doubt about some of the methodology as well as the material upon which rests such scientific, as distinguished from intuitive, knowledge of the matter as may be said to exist at this time. The work of Beeton and Pearson rests upon the assumption that if there is found to be a sensible degree of correlation between pairs of relatives in respect of age at death, the inheritance of the characteristic will have been demonstrated. But modern genetical theory plainly suggests that caution is needed here. Normal genetic situations are well known and understood, such as, for example, the blue Andalusian fowl type of inheritance,[11] in which, if nothing whatever was known about the genetic mechanism involved, a biometric study of somatic characters only, by the correlational method, would not be likely to add to our useful knowledge, and might easily lead to quite erroneous deductions.[12] Nothing whatever is really known of any genic mechanism having to do with longevity in man. While, in the complete absence of such knowledge, it is doubtless fair to say that a sensible correlation between near relatives in respect of age at death, creates a presumptive probability that there exists actually some degree of inheritance of the characteristic of the human organism upon which different degrees of longevity depend and of which they are expressions, any more positive conclusion than this would scarcely seem warranted from such premises. If, however, such correlations come out, when derived from more adequate material, to be sensibly equal to zero, the case will be still farther removed from a complete scientific demonstration of the inheritance of duration of life.

In this laboratory we have studied a somewhat more extensive collection of genealogical material than has hitherto been brought to bear upon the problem. Without any selection whatever, except for the omission of families in the later recorded generations in which the vital records were necessarily incomplete because sufficient time had not elapsed to complete them, we have extracted all the data from five American genealogical works, all of a relatively high standard of quality in such literature. These will be referred to as the Whitney,[13] the Hyde,[14]

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[11] Cf. Punnett, R. C., Mendelism. London (Macmillan), 1927. Pp. 64-66; 169; 204. Bateson, W., Mendel's Principles of Heredity. Cambridge (University Press), 1930. Pp. 51-53.

[12] See also in this connection, Pearl, R. Requirements of a proof that natural selection has altered a race. *Scientia*, Vol. 47, pp. 175-186, 1930.

the Spooner,[15] and the Smedley[16] families, and the Durham Town History.[17]

Table 1 gives some illustrative results derived from this material. Evidence as to the substantial actuarial normality of this material, taken as a whole, has been presented elsewhere.[18] It is there shown that life tables for fathers and for mothers from this material both give survivorship distributions at ages which agree closely in their *form* with comparable male and female survivorship distributions from Glover's[19] life tables, and differ from them in somewhat greater absolute values in some parts of the tables by only such amounts as might reasonably be expected from the fact that parents are a selected class.[20]

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[15] Phoenix, S. W. The Whitney Family of Connecticut and its Affiliations: being an Attempt to Trace the Descendants, as well in the Female as the Male lines, of Henry Whitney, from 1649 to 1878; to which is Prefixed some Accounts of the Whitneys of England. New York (privately printed), 1878. 3 vols. Pp. xxii + 2740.

[16] Walworth, R. H. Hyde Genealogy; or the Descendants in the Female as well as in the Male lines from William Hyde, of Norwich, with their places of residence, and dates of birth, marriages, etc., and other particulars of them and their families and ancestry. Albany (Munsell), 1864. 2 vols.

[17] Spooner, T. Records of William Spooner, of Plymouth, Mass., and his descendants. Cincinnati, 1883. Pp. 694.

[18] Cope, G. Genealogy of the Smedley Family descended from George and Sarah Smedley, settlers in Chester County, Penna., with brief notices of other families of the name, and abstracts of early English wills. Lancaster, Pa. (Wickersham Printing Co.), 1901. Pp. xi + 1000.

[17] Stackpole, E. S., and Meserve, W. S. History of the Town of Durham, New Hampshire (Oyster River Plantation) with Genealogical Notes. Concord, N. H. (Rumford Press), 1913. Pp. Vol. I, vii + 436; Vol. II, 502.

[18] Pearl, R. Alcohol and Longevity. New York (Alfred A. Knopf), 1926. Pp. 131-138 *passim*.

[19] Glover, J. W. United States Life Tables 1890, 1901, 1910, and 1901-1910. Washington (Bureau of the Census), 1921.

[20] For other evidence as to the relation of parenthood to longevity see Gould, A. The longevity of grandparents. *Eugenical News*, Vol. XII, pp. 166-171; 175-178, 1927. The superiority of married persons over single in respect of duration of life was pointed out many years ago by Farr (Influence of marriage on the mortality of the French people. *Trans. Nat. Assoc. for the Prom. of Soc. Sci.*, 1858, pp. 504-512. Ref. from Farr's "Vital Statistics," pp. 438-441, where the essential parts of the original are reproduced), and also by Stark (cf. *Med. Times and Gaz.*, 1867 (i), p. 227) although Stark somewhat unwarrantably generalized from his objective results.

The first section (A) of Table 1 gives the correlation, in respect of age at death, between all fathers and all sons, regardless of the ages at which the latter died (i. e., whether as adults or minors), for each separate family group and for all such groups together. The other two sections of the table (B and C) present the correlations between fathers and their *adult* sons and daughters respectively, for the whole material taken together; and the correlation between fathers and each of their sons and daughters separately (up to the seventh) by birth order of the latter; I, signifying first-born son (or daughter), II, the second-born, and so on. Offspring seventh and above in birth order are grouped together, in the case of both sons and daughters.

TABLE 1

Correlations between (a) Father's Age at Death and (b) Offspring's Age at Death. (Genealogical Data)

GROUPS CORRELATED	N	COEFFICIENT OF CORRELATION *r*
A. All fathers and all sons in		
1. Whitney Family	2,029	+ .022 ± .015
2. Hyde Family	857	+ .128 ± .023
3. Spooner Family	231	+ .077 ± .044
4. Smedley Family	1,165	— .064 ± .020
5. Durham Town History	999	+ .056 ± .022
6. All families (1-5) together	5,281	+ .030 ± .009
B. Fathers and sons by birth order of sons. All families (1-5) together.		
1. Fathers and all adult sons	4,407	+ .061 ± .010
2. Father and Son I	843	+ .046 ± .023
3. Father and Son II	775	+ .080 ± .024
4. Father and Son III	714	+ .013 ± .025
5. Father and Son IV	602	+ .057 ± .027
6. Father and Son V	511	— .005 ± .030
7. Father and Son VI	435	— .027 ± .032
8. Father and Son VII and over	1,073	— .002 ± .021
C. Fathers and daughters by birth order of daughters. All families (1-5) together.		
1. Fathers and all adult daughters	3,689	+ .047 ± .011
2. Father and Daughter I	669	+ .044 ± .026
3. Father and Daughter II	671	+ .044 ± .026
4. Father and Daughter III	597	+ .087 ± .027
5. Father and Daughter IV	530	— .022 ± .029
6. Father and Daughter V	484	+ .062 ± .031
7. Father and Daughter VI	394	+ .129 ± .033
8. Father and Daughter VII and over	918	— .012 ± .022

The following comments may be made upon the observations recorded in Table 1:

None of the coefficients deviates much from zero in absolute value. Of the 22 coefficients tabled only three are as much as four times their respective probable errors. Only eight are as much as three times their probable errors. They are not in unanimous agreement as to sign, though more are positive than negative (16+; 6—). There appear to be no significant differences between the coefficients for the different families; nor between those for the children of different birth orders; nor between those for adult children and all children. Altogether it seems reasonably evident that the father-offspring correlations for this fairly extensive sample of genealogical data would suggest, if no other knowledge or experience were available, that the age at death of one's father either was worth nothing at all in predicting one's own probable age at death, or at the best was worth so little that it might be quite safely left out of all practical consideration.

But the common sense and the common observation of mankind, as well as the practical experience of insurance companies, indicate that the age of his father at death is not so negligible a factor in predicting an individual's own probable length of life as these correlation observations appear to indicate. Furthermore Bell [21] picked out from the Hyde Family (No. 2 of Section A of Table 1 *supra*) with which he worked, 128 cases in which the average age at death of the offspring of parents both dying under 60 years of age was 32.8 years, whereas in 184 cases in which both parents died at ages over 80 years the average age at death of the offspring was 52.7 years.

The interpretation of the *biological* meaning of a correlation coefficient is rarely completely clear and unequivocal, and in this case it is particularly obscure. Genealogical material is known to have peculiarities arising from the methods by which it is accumulated. It probably also has inherent biological peculiarities.[22] Perhaps the correlation method *per se* is not well adapted to deal effectively with the problem of the inheritance of duration of life; or, to put it in another and possibly better way, perhaps we do not quite understand how to formulate the problem so as to permit a clear and unequivocal answer to be reached by the correlational methodology. In any case, after long study of the correlation results, it seemed desirable to turn to another mode of attack,

[21] Bell, A. G., *loc. cit.*, p. 30.

[22] See Gould, *loc. cit. supra* in this connection.

using material which had been collected under critical supervision and control. To the new results so attained we may now turn.

II

Some years ago there was collected under my direction [23] rather elaborate and detailed data regarding something over 100,000 individuals mostly resident in or near Baltimore. These Family History Records, as they are called in the laboratory, consist basically of extensive pedigrees, collected by a staff of field workers especially trained for this work. In most instances these field workers had, in addition, previous experience in some form of social work, as well as general biological training in college or university.

These field workers visited the families of individuals whom it was desired to investigate, and got by personal interview the information to be set down in the histories. Proceeding in the matter in an entirely objective way and without preconceptions, it was felt to be essential to get the most elaborate and critically exact records possible about the environmental situation, the habits of life, the health history, the racial stock, anthropological characteristics, etc., of all the members of the family group.

Every critical safeguard of the accuracy of the ultimate individual records that we were able to think of was thrown around this work. No material fact was entered in a history finally until it had been corroborated by the independent testimony of at least two persons acquainted with the individuals in question. The material has all been taken from one socially and economically fairly homogeneous group of the population of the City of Baltimore and Maryland; namely, what may roughly be called workingmen's [24] families, in the sense that they were people

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[23] With financial aid, which is hereby gratefully acknowledged, from the American Tuberculosis Association, The Russell Sage Foundation, and the Commonwealth Fund.

[24] This designation is inadequate and has led to misunderstanding of the material. It has been assumed by some commentators on earlier work with the material that we were dealing with slum dwellers, in the lowest walks of life and condition of wretchedness. This is a quite erroneous idea. The people included in the Family History Records belong mainly in the skilled labor, artisan, small shop keeper, clerical, and factory worker class. Relatively few fall in the social and economic classes lower than these, and such as do are roughly counterbalanced in number by those in higher classes. The difficulty, of course, is that the American language is not rich in terms sharply and briefly delimiting social and economic classes, perhaps chiefly because social classes themselves are not very sharply delimited in American urban life.

who had to work for a living. By the employment of field workers speaking a variety of foreign languages it was possible to get representation in the histories of the different foreign race stocks in about the proportion that they are represented in the total population of the City of Baltimore. In this way there was no racial discrimination in the study. The only exception to this is that the negro was not included, owing to the difficulty—indeed impossibility—of getting reliable genealogical information about negro families.

These Family History Records have been utilized for various investigations which have been made in my laboratory,[25] and their characteristics are well understood. While the material is excellent for the present purpose in certain respects, it has defects in others. As in all extensive collections of human records there is a tendency for stated ages to concentrate on the multiples of 5. This tendency is not so marked in this material as it is in official census returns, but still there is some evidence of it. One of the greatest defects in the records is missing information—missing because it was impossible to obtain. Thus in a sibship of 9 persons the records may be complete and accurate for 8, and all that is known of the ninth member is that it was a male who left home, "went out West," and has not been heard of since. The net effect of this entirely human phenomenon is that the whole sibship becomes useless for such investigations as this, because it is incomplete. Our experience is that the wastage from such cases as these in any collection of family records is considerable. Furthermore, in material collected in the way this was, the data available in the end are unevenly distributed as to generations. There are great numbers of records of persons in middle life. Their children, and their parents are also abundantly and completely recorded. But heavy gaps begin to appear in the records of the grandparental generation, and still more in the great-grandparental (relative, that is, to the persons in middle life). There seems no escape from this difficulty. Ask anyone merely to name and give the ages at death of his great-grandparents. Few indeed are those who can give this information without searching the family archives. And there are so few people, statistically speaking, who have any family archives to search.

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[25] Particularly Pearl, R. Alcohol and Longevity, *cit. supra*, in which will be found a detailed account of the material; The constitutional factor in breakdown of the respiratory system. *Annals of Eugenics*, Vol. 2, pp. 1-24, 1927; The age at death of the parents of the tuberculous and the cancerous. *Amer. Jour. Hyg.*, Vol. 3, pp. 71-89, 1923.

Wherever there are gaps in the records of the sorts just discussed the cautious investigator will always have in mind the possibility that there may be a selective element involved. There is, in short, the possibility that the individuals who are missing in the records may be a differentiated lot, in respect of the character under investigation. We have never encountered anything to make us suspect that this was so in the Family History Records, but the possibility must be kept in mind.

While our Family History Records are far from perfect, I am certain that they are more reliable, complete, and comprehensive than any published genealogical records with which I am acquainted.

From these Family History Records all the pertinent data for the problems discussed below were extracted, without selection, according to the following division of labor:

Cases 1 to 23 inclusive, by Raymond Pearl
Cases 51 to 89 inclusive, by John R. Miner
Cases 101 to 128 inclusive, by Ruth DeWitt Pearl
Cases 151 to 167 inclusive, by Takeo Imai
Cases 201, 203, 204, by I-chin Yuan.

As the Family History Records in total include 437 cases, it will be seen that this preliminary report includes only about 25 per cent of all the available *cases*.[26] The work is being continued, and a final report will include all the material.

III

The first question put to the material was of this form: Are the life tables of *parents* of children dying within specified age limits, differentiated, one from another, and if so, in what way relative to the ages at death of their children? For example, suppose a life table to be constructed for the *fathers* of all children who died under 5 years, and suppose another life table to be constructed in precisely the same way for the *fathers* of all children [27] dead (or living) at ages of 80 and above. Will these two life tables be identical with, or different from, each other, having regard to such fluctuations as might reasonably be expected from sampling?

If it turns out that the two life tables are different, the one from the other, a probability will then be established that the fathers of children who died in infancy and early childhood form a group of men differentiated from fathers of children [27] living to 80 years of age

[26] It should be understood that each "case" may contain anything from 50 to more than 5,000 *individuals*.

and above, in respect at least of their order of dying, or, more simply, of their longevity.

In order to get an answer to the first question the procedure was as follows: Data sheets were first prepared for age groups according to the children's age at death, and for all the parental and grandparental relationships. The limits of the children's age at death classes were: Under 5, 5 to 24, 25 to 49, 50 to 69, 70 to 79, 80 and over (living or dead). In all classes up to the last only the dead children [27] were considered, but whether a child [27] is dead or alive at ages of 80 and above it is clear that such a person has achieved great length of days, and nothing can subsequently happen which can possibly alter that fact.

Frequency distributions of the ages of each of the parents and grandparents (father, mother, father's father, father's mother, mother's father, mother's mother) separately tabled for the living and for the dead, were then formed for each of the age-at-death classes of the children noted above. From these distributions life tables were constructed [28] by standard actuarial methods. Each of the specified ancestors was entered in the appropriate table once for each of his dead progeny. Thus if a living father aged 35 has had three children die at ages of 1 year, 2 years, and 3 years respectively, the father will be entered in the "Children dead under 5—Father living," distribution, three times at age 35. Again, if a father has had no children die, he will not appear in any table, unless at least one of his children has attained the age of 80. The effect of the former point is to weight ancestors according to a combination of their fertility and the mortality of their progeny.[29] The effect of the latter is simply to leave out of

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[27] In the kinship sense of the word.

[28] Without graduation in the case of the preliminary trials here reported.

[29] The varying size of offspring sibships is one of the many difficulties which always confront one in any attempt to deal statistically with the problem of inheritance in man. The method outlined above of dealing with this particular aspect of an extremely complex problem is only one of several possible methods, all of which must ultimately be tried out before the final definitive analysis of the problem can be regarded as completed. Our purpose is to try to analyze the problem bit by bit, and after careful consideration it was decided to adopt the method described for the first trial. As a matter of fact this method, in so far as its effect is not neutral with large numbers, tends to minimize any apparent inheritance of longevity.

account for the moment a good deal of available pertinent material, which, however, will be taken into account in other angles of attack on the problem—by the postulation of other questions, in short.

We have chosen in this preliminary report to present the essential results indicated by the tables in the form of expectations of life (mean-after-lifetimes) at ages. In the final report other life table functions will also be tabled. The results are shown in Figs. 1 to 6 inclusive. All of these diagrams are constructed on the same plan. The heights of the rectangular bars are equal to the expectations of life in years given in figures at the top of each bar. The expectations are given for the four ages 20, 40, 60, and 80, indicated on the abscissal axis. The solid bars are in each case for the *lower* of the groups compared on the basis of the children's age at death; the cross-hatched bars for the *higher* of these groups.

On account of the relatively small amount of the material so far extracted from the original records we have combined the children's age-at-death groups for the first comparisons into two broad groups, *viz.*: (1) children dead at ages under 50 years, and (2) children dead at ages of 50 years or over.

The magnitude of the present material may be indicated as follows:

Life Table	*Life Years Exposure to Risk*
Fathers of children dying under 50 years of age	91,972.5
Fathers of children dying 50 and over years of age	17,595.0
Mothers of children dying under 50 years of age	92,362.5
Mothers of children dying 50 and over years of age	18,065.0
Grandfathers [30] of grandchildren dying under 50 years of age	96,935.0
Grandfathers [30] of grandchildren dying 50 and over years of age	1,867.5
Grandmothers [30] of grandchildren dying under 50 years of age	98,225.0
Grandmothers [30] of grandchildren dying 50 and over years of age	2,060.0

[30] Both paternal and maternal combined. The experience embodied in this sample for grandparents of persons dying at ages of 50 and over is small and the results for these groups must, on that account, be viewed with great caution and reserve.

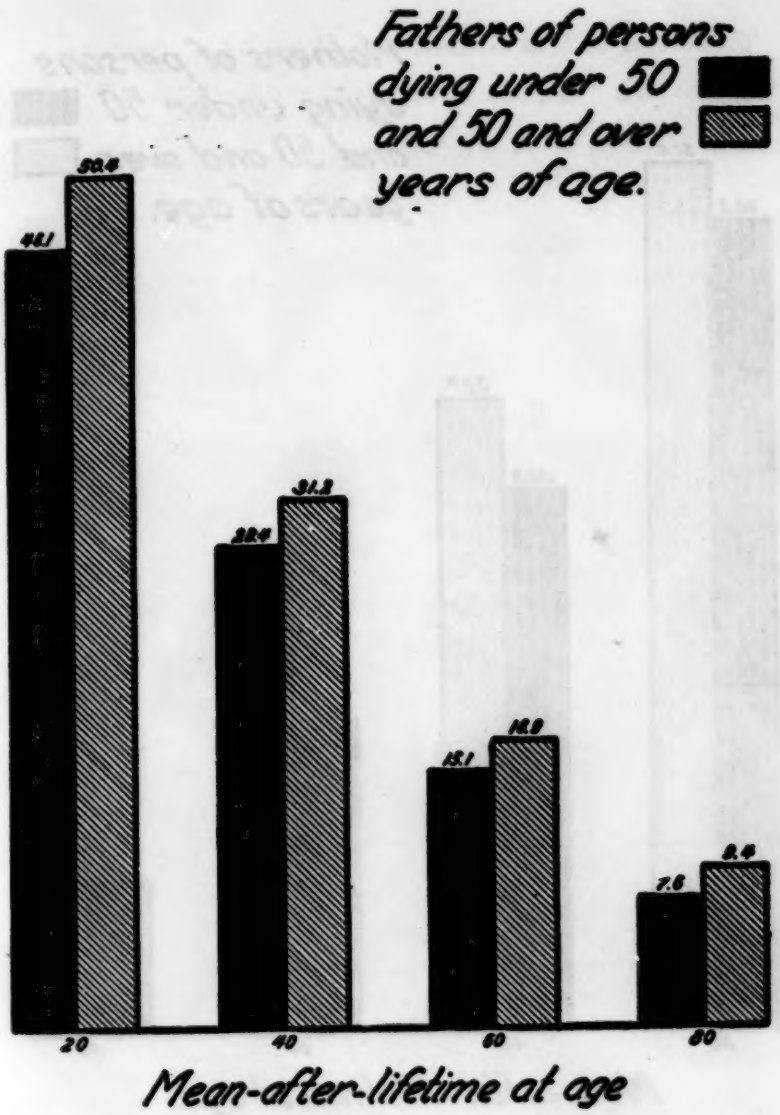

FIG. 1. EXPECTATIONS OF LIFE IN YEARS (MEAN-AFTER-LIFETIME) AT AGES 20, 40, 60, AND 80, OF FATHERS OF CHILDREN DYING (A) UNDER 50 YEARS OF AGE (SOLID BARS), AND (B) 50 AND OVER YEARS OF AGE (CROSS-HATCHED BARS)

FIG. 2. LIKE FIG. 1, BUT FOR MOTHERS

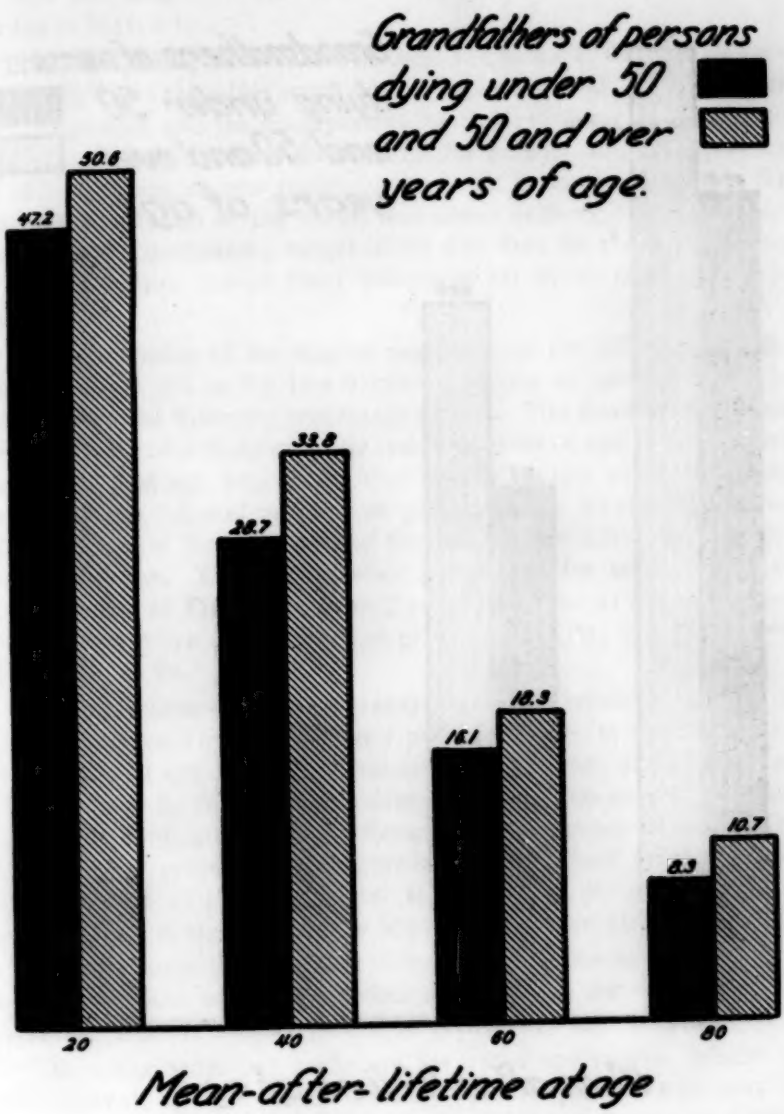

FIG. 3. LIKE FIG. 1, BUT FOR GRANDFATHERS (PATERNAL AND MATERNAL COMBINED)

FIG. 4. LIKE FIG. 1, BUT FOR GRANDMOTHERS (PATERNAL AND MATERNAL COMBINED)

The following comments may be made upon the observations recorded in Figs. 1 to 4:

The outstanding point shown in these life tables is that for all ages from 20 (where the tables start) on, the expectations of life are *greater* for the parents, and the grandparents, of children dying at ages of 50 and over years of age than they are for the parents, and grandparents, of children dying at ages under 50 years. It is the uniformity in the direction (or sense) of the results that seems more significant and important in this preliminary sample of the data than the absolute amounts of the differences, though these differences are by no means slight in amount.

An appreciation of the relative magnitude of the differences in the expectation of life in the two compared groups of ancestors may be gained from the following percentage figures. The mean-after-lifetime of *fathers* of children dying at 50 and over years of age is about 9 per cent greater at age 20; 10 per cent greater at age 40; 12 per cent greater at age 60; and 24 per cent greater at age 80 than the mean-after-lifetimes at the same ages of the fathers of children dying under 50 years of age. The corresponding percentages for *mothers,* derived from the data of Fig. 2, are about 7 per cent greater at age 20; 17 per cent greater at age 40; 22 per cent greater at age 60; and 28 per cent greater at age 80.

The mean-after-lifetime of *grandfathers* of grandchildren dying at 50 and over years of age is about 7 per cent greater at age 20; 18 per cent greater at age 40; 14 per cent greater at age 60; and 29 per cent greater at age 80 than the mean-after-lifetime at the same ages of the grandfathers of grandchildren dying under 50 years of age. The corresponding percentages for *grandmothers,* derived from the data of Fig. 4, are 18 per cent greater at age 20; 35 per cent greater at age 40; 42 per cent greater at age 60; and 59 per cent greater at age 80.

It is to be expected that these differences, both absolute and relative, may be modified when all the data are available for analysis. The present figures are to be regarded as preliminary and tentative.

The comparisons just made are for broad age groups, relative to the children's ages at death. A more specific question may now be asked. Are the life tables of parents of children dying under 5 years of age differentiated, and if so in what manner and degree, from the life tables of the parents of children dying (or living) at 80 or more years of age?

The answer given by the present sample of material from our Family History Records is shown in Figs. 5 and 6. These diagrams are constructed on the same plan as Figs. 1 to 4. The magnitude of the experience embodied in them is as follows:

Life Table	*Life Years Exposure to Risk*
Fathers of children dying under 5 years of age	53,432.5
Fathers of children dying at 80 and over years of age	2,915.0
Mothers of children dying under 5 years of age	53,570.0
Mothers of children dying at 80 and over years of age	3,557.5

These observations indicate that when the two groups of parents compared are widely separated in respect of the ages at death of their children, as is the case in Figs. 5 and 6, their own life tables are markedly differentiated, the one from the other. At every age from 20 on, at which age the life tables start, the mean-after-lifetime of the parents of the extremely long-lived children is *greater* than that of the parents of the extremely short-lived children and by considerable amounts.

It is well at this point again to make quite clear how these results have been reached, to the end that their meaning may be the better understood and judged. There has been picked out of the material used *every* single extremely short-lived child (who lived less than 5 years), regardless of all other considerations except this single one of brevity of life. Life tables have then been constructed for the fathers and for the mothers of these short-lived children. These tables give definite information as to whether these parents, taken as a lot, are characterized by short life or long life. Next there has been picked out of the material *every* single person of great old age (who lived to 80 years of age or above), regardless of any other consideration except that of great longevity. Just as before, life tables have been calculated for the parents of these long-lived children.

When these two sets of life tables are compared it is plain that, so far as this present sample of material is concerned, the long-lived children had parents much longer-lived than the parents of the short-lived children. This result is clear, but must be regarded for the present as preliminary and tentative, and subject to the test of larger experience.

The magnitude of the differences exhibited in Figs. 5 and 6 may be stated in relative terms as follows: The mean-after-lifetime of the

FIG. 5. EXPECTATION OF LIFE IN YEARS (MEAN-AFTER-LIFETIME) AT AGES 20, 40, 60, AND 80, OF FATHERS OF CHILDREN DYING (A) UNDER 5 YEARS OF AGE (SOLID BARS), AND (B) 80 AND OVER YEARS OF AGE (CROSS-HATCHED BARS)

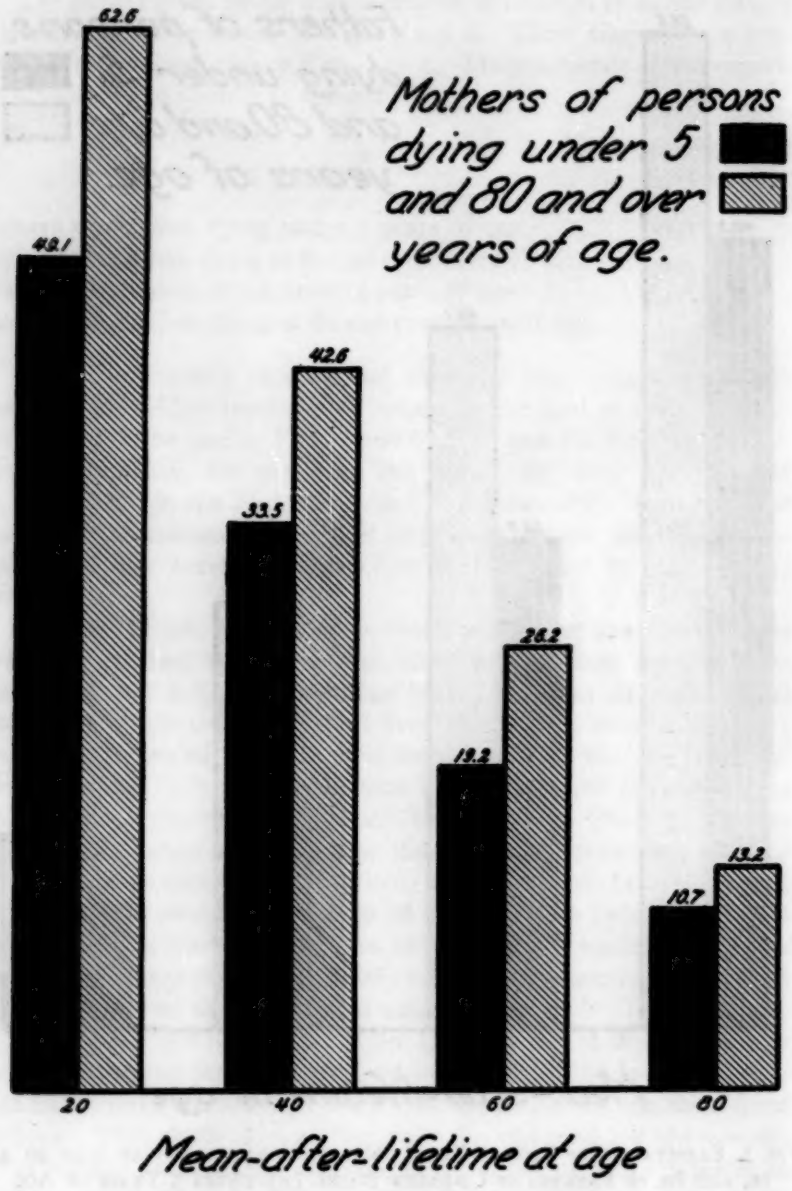

FIG. 6. LIKE FIG. 5, BUT FOR MOTHERS

fathers of children dying (or living) at ages of 80 and over is about 26 per cent greater at age 20; 43 per cent greater at age 40; 75 per cent greater at age 60; and 58 per cent greater at age 80, than the mean-after-lifetimes at the same ages of the fathers of children dying under 5. Similarly the mean-after-lifetime of the *mothers* of children dying (or living) at ages of 80 and over is about 27 per cent greater at age 20; 27 per cent greater at age 40; 36 per cent greater at age 60; and 23 per cent greater at age 80 than the mean-after-lifetimes at the same ages of the mothers of children dying under 5.

IV

Having reached this stage in this study, and having seen that the present sample of data indicates that parents, and grandparents, appear to fall into differentiated groups in respect of longevity, according to the ages at which their children (or grandchildren) died, we may proceed to put to the material another question. This second general question may be framed as follows:

Are the life tables of all children, taken without selection or omission, of parents dying at specified ages, differentiated, one from another, and if so in what way, relative to the ages at death of their parents?

For example, suppose a life table to be constructed by standard actuarial methods for the sons of fathers dying under 50 years of age; suppose another similar life table to be constructed for sons of fathers dying between 50 and 79 years of age; and finally suppose a third life table to be constructed for sons of fathers dying (or living) at 80 or more years of age. Will these three life tables be identical with, or different from, each other, and if so, in what manner?

This question is the reverse of the one discussed in the preceding section. It is now asked whether the age at death of a parent is a factor of any importance in estimating the probable duration of life of the offspring. If there is a significant degree of inheritance of longevity the answers to the two questions should be the same in sense. The results will be a check upon each other.

Using the same case histories as before (listed on p. 254 *supra*) the same persons [21] extracted the material from which to construct the life tables necessary to get an answer to our second question. Only completed offspring sibships were used. By a completed sibship is to be

[21] With the exception of Dr. Yuan, whose histories were extracted by Miss Pearl in the second study.

understood a group of siblings produced by a mating from which no further offspring can be produced, by reason either of the death of one or both parents within the period of reproduction, or because the mother has passed the child-bearing age. Furthermore all children in the sibship must have records of sex and age. All completed offspring sibships in the listed cases were used, without selection, provided one or both parents had died (or was living at 80 or more years of age). In constructing the life tables both living and dead children were included.

In this preliminary report only one set of results can be presented. These relate to the "sons of fathers" tables. The magnitude of the material may be indicated as follows:

Life Table	*Life Years Exposure to Risk*
Sons of fathers dying under 50 years of age	14,330
Sons of fathers dying between 50 and 79 years of age	39,645
Sons of fathers dying (or living) at 80 or more years of age	9,835

The results are shown in Fig. 7, which is constructed on the same plan as the earlier diagrams. The numbers at the top of each bar are the mean-after-lifetime (expectation of life) in years, at the ages indicated on the abscissal axis.

The observations recorded in Fig. 7 indicate plainly that the mean-after-lifetime of the sons, in the material analyzed, was lowest when the fathers died young (under 50); was higher when the fathers died at higher ages (50-79 inclusive); and was highest when the fathers lived to the age of 80 and over. The answer to our second question is thus in agreement with the answer to our first. Life tables for "daughters of fathers," for "sons of mothers," and for "daughters of mothers," give results corresponding in principle to those shown in Fig. 7, with, of course, some numerical differences and irregularities to be expected from sampling.

The relative magnitudes of the differences exhibited in Fig. 7 are as follows: The mean-after-lifetime of the sons of fathers dying (or living) at ages of 80 and over is about 13 per cent greater at birth (age 0) than that of the sons of fathers dying between 50 and 79 years of age, and 22 per cent greater than that of the sons of fathers dying under 50 years of age. The corresponding percentages for the expectation of life of the same groups at age 20 are 3 and 12 per cent; at

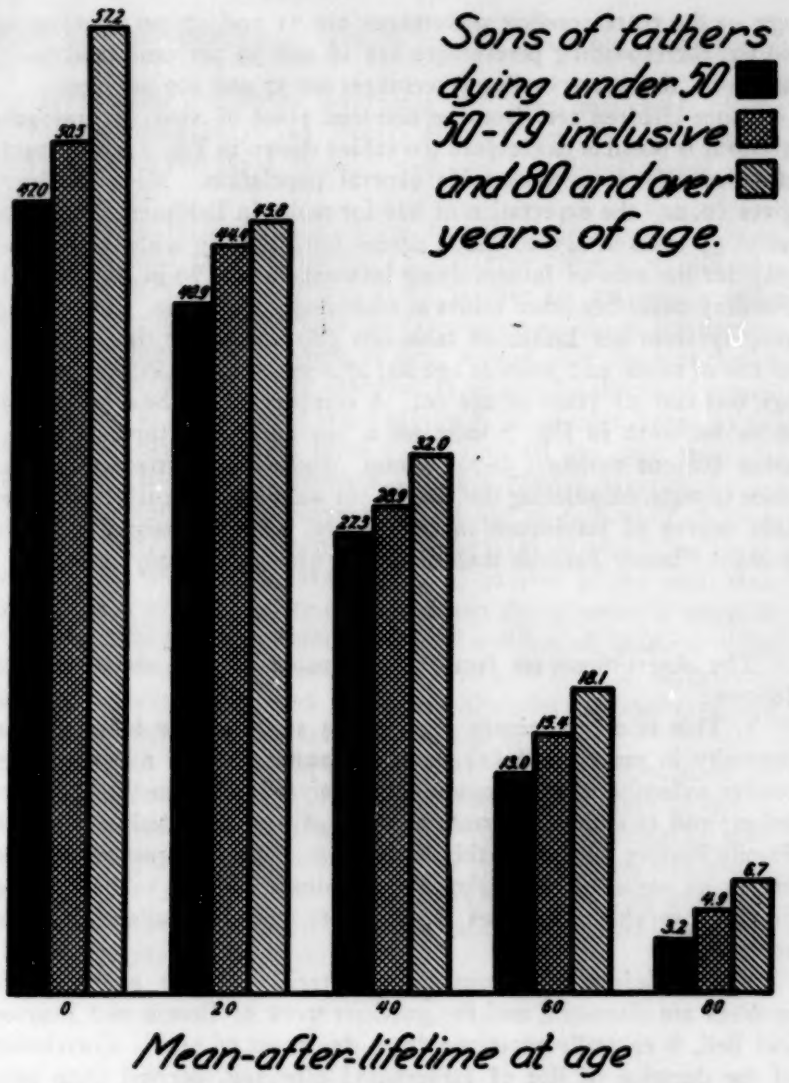

FIG. 7. EXPECTATION OF LIFE IN YEARS (MEAN-AFTER-LIFETIME) AT AGES 0 (BIRTH), 20, 40, 60, AND 80 YEARS OF SONS OF FATHERS DYING (A) UNDER 50 YEARS OF AGE (SOLID BARS); (B) BETWEEN 50 AND 79 YEARS OF AGE (DOUBLE CROSS-HATCHED BARS); AND (C) 80 AND OVER YEARS OF AGE (SINGLE CROSS-HATCHED BARS)

age 40 the corresponding percentages are 11 and 17 per cent; at age 60 the corresponding percentages are 18 and 39 per cent; and finally, at age 80, the corresponding percentages are 37 and 109 per cent.

Since children are, from an actuarial point of view, an unselected group it is possible to compare the values shown in Fig. 7 with expectations of life in a comparable general population. Miss Foudray [22] gives (p. 24) the expectation of life for males in Baltimore, 1919-1920, as 51.55 years at age 0, which agrees fairly closely with the value of 50.5 for the sons of fathers dying between 50 and 79 in Fig. 7. Miss Foudray tables her other values at midquinquennial ages. Interpolating roughly from her Baltimore table one gets values for the expectation of life of about 42.7 years at age 20; 27.2 years at age 40; 13.7 years at age 60; and 5.1 years at age 80. A comparison of these values with those set forth in Fig. 7 indicates a fair agreement throughout with those for our middle (50-79) group. Indeed the agreement appears close enough, considering the size of our samples, to justify a considerable degree of confidence in the general actuarial normality of the Family History Records material upon which this study is based.

V

The observations set forth in this paper may be summarized as follows:

1. This is a preliminary report of a study of the inheritance of longevity in man, based (a) upon an examination of a new and extensive collection of genealogical data, analyzed by the method of correlation; and (b) upon an actuarial study of new data derived from the Family History Records of this department. All of the results presented are to be regarded as preliminary and tentative and subject to confirmation or alteration when the complete material available has been analyzed.

2. The defects of genealogical material for the study of the problem are discussed, and the previous work of Beeton and Pearson, and Bell, is critically examined from this point of view. Correlations of the duration of life of fathers and offspring, derived from more extensive genealogical material than has hitherto been analyzed in this way, are not, as a whole, sensibly different from zero, and could not, if they stood alone, be regarded as giving evidence of any sensible or

[22] Foudray, Elbertie. United States Abridged Life Tables 1919-1920. Washington, Bureau of the Census, 1923. Pp. 84.

regular parental inheritance relative to this character, duration of life.

3. Life tables embodying something less than one-fourth of the total material available in our Family History Records are discussed. These life tables rest upon, in total, well over a half-million life years exposure to risk.

4. These life tables indicate: First, that the expectation of life of the *parents* (either father or mother) of children dying at 50 and over years of age is, at all ages from 20 on, *greater,* by amounts varying from about 7 to 28 per cent, than the expectation of life of the parents of children dying under 50 years of age; second, that the expectation of life of *grandparents* (either grandfather or grandmother) of grandchildren dying at 50 and over years of age is, at all ages, from 20 on, *greater,* by amounts varying from about 7 to 59 per cent, than the expectation of life of grandparents of grandchildren dying under 50 years of age; third, that the expectation of life of *fathers* of children dying (or living) at 80 and over years of age is, at all ages from 20 on, *greater,* by amounts varying from about 26 to over 50 per cent, than the expectation of life of *fathers* of children dying under 5 years of age; fourth, that the expectation of life of *mothers* of children dying (or living) at 80 and over years of age is, at all ages from 20 on, *greater,* by amounts varying from 23 to 36 per cent, than the expectation of life of mothers of children dying under 5 years of age.

5. These life table studies also indicate that the expectation of life of *sons* of fathers dying (or living) at 80 and over years of age, is *greater* at all ages from birth on, than the expectation of life of sons of fathers dying at ages between 50 and 79 years inclusive, and is *still greater* than the expectation of life of sons of fathers dying under 50 years of age. These differences are regular and considerable in amount.

The investigation is being continued.

A STUDY OF LIKE-SEXED TWINS

PART II. THEIR HEALTH AND DISEASE RECORDS

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A. INTRODUCTION

Up to the present time the majority of scientific reports upon twins have dealt with small samples, or with individual pairs selected on the basis of some outstanding peculiarity. Where large numbers of cases have been available, it has been customary to restrict the study to cross-section analyses, without attempting to make use of developmental records. The following report deals in a preliminary manner with intra-pair differences in the health and disease histories of 161 pairs of twins, classified as "identical," "fraternal," and "undetermined" (Table 1). The average ages of these groups are given in Tables 2 and 3. This group is part of a larger sample of twins located in an Institute of Child Welfare survey, and includes all of the twins of school age whose mothers could be reached in home interviews. The general characteristics of the sample, the technique of the interview, and the criteria for classification, have been described in a previous article (4). The families are predominantly native white, of north European descent; their social status distribution, on the basis of the Berkeley Social Rating Scale, is adequately representative for the four California cities from which the main twin sample was drawn.

TABLE 1

Number and Sex of Twins Studied

TYPE	MALE	FEMALE	COMBINED
Identical	25	44	69
Fraternal	34	38	72
Undetermined	11	9	20
Totals	70	91	161

TABLE 2

Ages of Twins (In Years)

TYPE	NO.	RANGE	MEDIAN	MEAN	S. D.
Identical	69	5.83 to 21.42	13.83 ± .37	13.70 ± .30	3.69 ± .21
Fraternal	72	5.83 to 20.18	11.44 ± .31	11.46 ± .25	3.13 ± .18
Undetermined	20	6.00 to 19.00	10.60 ± .59	10.98 ± .46	3.19 ± .33

TABLE 3

Distribution of Sample by Ages

	AGE: IN YEARS				
TYPE	5 TO 8.9	9 TO 12.9	13 TO 16.9	17 AND OVER	TOTALS
Identical	12	15	27	15	69
Fraternal	20	32	16	4	72
Undetermined	7	9	3	1	20
Totals	39	56	46	20	161

B. THE DATA

1. *Diseases*

The disease record of each twin was obtained through an interview with the mother. She was questioned specifically about the incidence of each of the more common communicable diseases[1] and her answer recorded on a printed record form. With the data secured in such a manner the following factors must be considered:

(a) The mother may forget whether or not a child has had a certain disease. This would occur more often to mothers of large families or where several years have elapsed since the attack. Fortunately for this study such an error would not often occur where a disease attacked but one member of a pair of twins, as such an unusual occurrence would seldom be forgotten.

(b) There might be errors of diagnosis. However, in these communities most of the communicable diseases considered in the study are subject to stringent quarantine rules. Local health officials are responsible for their enforcement and errors of diagnosis would seldom occur.

As may be noted from Table 4, the incidence of particular diseases varied considerably,[2] with measles, whooping cough, and chicken pox

[1] The data on influenza, smallpox and diphtheria were obtained in answer to the questions on "other diseases."

[2] In three cases both members of a pair had a certain disease, but at different ages. These cases have not been included in the "difference in incidence" data.

heading the list. As a measure of the prevalence of influenza the percentages are probably not very reliable because of the difficulty of distinguishing this disease from the common "cold." However, the figures for the different groups of twins should be comparable. The percentage of children vaccinated would be a factor in determining the incidence of smallpox. Unfortunately, no data on this matter were secured from the mothers.

The incidence figures for identicals and fraternals are closely similar. For all the "communicable" diseases taken together, the identicals had an average incidence of 3.23, and the fraternals of 3.10. The excess of diseases among the identicals is probably explained by their greater average age. In the case of the so-called "constitutional" diseases, information of considerable interest was obtained, but the data were too restricted to be treated statistically, and there may also be some doubt as to the reliability of the diagnoses. In the case of the contagious children's diseases, however, (particularly those subject to quarantine) we have no reason to suspect the accuracy or the completeness of the data.

TABLE 4

The Incidence of Specific Communicable Diseases as Shown by Percentage of Individuals Having Had the Disease

	TOTAL SAMPLE	IDENTICALS	FRATERNALS	UNDETERMINED
Number:	322	138	144	40
Disease	%	%	%	%
Measles	82.3	86.2	80.1	75.0
Whooping cough	71.1	69.6	79.2	47.5
Chicken pox	68.6	63.8	69.4	85.0
Mumps	36.3	34.1	39.6	32.5
Scarlet fever	13.0	15.2	8.3	22.5
German measles	12.1	21.7	5.6	2.5
Influenza	9.6	11.6	7.6	10.0
Smallpox (or varioloid)	6.8	9.4	6.3	0.0
Diphtheria	5.3	9.4	2.1	2.2
Pneumonia	4.7	2.2	6.9	5.0
Others (*)	4.3	6.5	4.8	0.0
Averages:	28.7	30.0	28.2	26.1

NOTE: (*)—These included spinal meningitis, pink eye, laryngitis, typhoid, erysipilas, and dysentery. Most of these were single cases.

Similarity of disease records may be a significant factor in the study of twin or sibling resemblances in other traits. If twins have the same

disease more often than ordinary sibs, twin resemblance in intelligence, for example, may be heightened as compared with ordinary sib resemblance. In view of their close environmental association, it might on some grounds be expected that twin individuals would show a higher incidence of disease than children representing single births. Two children have a greater chance than one, of receiving infections from extra-familial contacts, and the transmission from one twin to the other may be very difficult to avoid. In this matter mothers appear to be somewhat fatalistic: when one of a pair contracts a children's disease, there is seldom any attempt to isolate his mate. However, data secured in this study do not indicate that twins on the whole have any more diseases than the single born.

TABLE 5

The Percentage of Individuals Having Had Certain Diseases
A Comparison of:

Twins with average age of 12.42 years,
Collins' "normal" children of 12.1 to 13.1 years, and
Terman's "Gifted" children with an average age of about 11.5

	PERCENTAGE OF INDIVIDUALS WHO HAVE HAD DISEASE		
DISEASE	*Collins' data*	*Terman's data*	*Twin sample*
Measles	88.2	87.2	82.3
Whooping cough	75.7	67.6	71.1
Chicken pox	50.3	61.1	68.6
Mumps	50.5	35.4	36.3
Scarlet fever	13.0	8.2	10.2
Influenza	Not given	19.3	9.6
Diphtheria	8.5	5.9	5.3
Pneumonia	Not given	4.6	4.7

Collins (1) has computed the age incidence of the more important communicable diseases of children. His study combines the data from various investigations in the United States and Canada and includes the disease histories of some thirty or forty thousand persons. He concludes that his figures give ". . . the mean incidence (of the diseases) for a considerable period of years under conditions such as exist in the more or less representative urban and rural communities where the data were collected." When his "norms" are compared with data for the twin sample (Table 5), we find that the rates are very similar.

Terman's (3) group of gifted children (about 650 individuals) were selected from cities in California, more than one-fifth of the number being located in the East Bay district from which our sample was taken. The average age of his group was about eleven and a half years. They usually came from superior homes. The data were secured from a questionnaire sent to the mother. Comparing these figures with ours (Table 5) we find that they agree even more closely than do those of Collins(1).

In this study, comparison of intra-pair differences in identical and fraternal twins has been employed in the analysis of hereditary factors in development. If a trait is highly subject to hereditary determination, we would expect to find it occurring with greater intra-pair differences among fraternals than among identicals. *Other things being equal,* the relative degree of hereditary influence in a series of traits can be appraised if these traits are placed in a rank-order according to the difference between the average intra-pair differences in identicals and in fraternals.

Siemens (2) has dismissed heredity as a factor in the incidence of contagious diseases of children, stating: "Among the non-hereditary maladies are the acutely contagious diseases which usually affect both twins at the same time. For example, in my material, I have never found a case where one twin has been attacked by measles while his mate has gone free." In the present sample, however, the contagious diseases[3] show intra-pair differences in 8.8 per cent of the identical pairs, and in 12.9 per cent of the fraternal pairs.[4] Stated otherwise, if an identical twin contracts a disease, his mate has 912 chances out of 1,000 of coming down with the same disease; if the twins are non-identical, the mate has 871 chances out of 1,000 of infection. The difference between these percentages is not fully reliable, being about two and a half times the PE of the difference.

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[3] In the data for the "constitutional diseases," the following cases were found in which both members of a pair suffered from the same disease: hives, 1 identical and 2 fraternal; croup, 1 identical and 1 undetermined; heart trouble, 1 identical; rheumatic fever, 1 fraternal.

[4] The difference in age should be considered when comparing the percentages of intra-pair differences in the groups. The older the children become the more diseases they have, and a greater number of intra-pair differences are to be expected. This is found to be the case in our data, when the number of intra-pair differences at each age are plotted on a scattergram. We may infer that the identicals and fraternals would show greater differences than are here reported, if the fraternals had averaged as high a chronological age as the identicals.

TABLE 6

Disease Incidence by Pairs

	NUMBER OF PAIRS IN WHICH BOTH WERE AFFECTED			NUMBER OF PAIRS SHOWING INTRA-PAIR DIFFERENCES IN INCIDENCE		
	Id.	*Fra.*	*Undet.*	*Id.*	*Fra.*	*Undet.*
Measles	59	56	15	1	4	0
Whooping cough	47	56	9	2	2	1
Chicken pox	43	48	17	2	4	0
Mumps	22	27	5	3	3	3
Scarlet fever	9	4	4	3	4	1
German measles	14	2	0	2	4	1
Influenza	7	5	3	2	1	0
Smallpox (or varioloid)	6	4	0	1	1	0
Diphtheria	6	1	0	1	1	1
Pneumonia	0	3	0	1	4	2
Others	3	2	0	1	3	0
Totals	216	208	52	19	31	9

Table 6 presents data for the diseases listed separately with the numbers of pairs having had each disease and also the cases in which a disease had affected only one member of a pair. When classified in this manner, the cases are too few to justify presentation in terms of percentages. In 11 classifications, intra-pair differences were in 6 cases more common among the fraternals, in one case more common among the identicals and in 4 cases no difference appeared. So far as this evidence goes, it appears to support the concept of hereditary factors in susceptibility to disease; providing, of course, that environmental intra-pair differences, which could have any possible significance for the present study, are presumed to be substantially equivalent for identicals and fraternals.[5]

It may be noted that the intra-pair differences in incidence occur in inverse ratio to the prevalence of a disease. This, of course, is to be expected, for a disease that strikes most children would be expected to affect both members of a twin pair. Susceptibility to measles, for example, is so widespread that hereditary differences in degrees of susceptibility are inadequate to manifest themselves in the incidence data. Such differences, however, might appear to a more marked degree in the severity of the disease, as indicated by febrile phenomena, duration

[5] A study is now being made of some of these environmental differences.

of illness, and specified after-effects.[8] First-hand objective data on these matters are lacking. The mothers, however, were asked the following question: "When the twins had the measles (or some other disease) were they both equally sick or did it seem to strike one child harder than the other? Did either child show any after-effects?" In measles 8 pairs of fraternals and 4 pairs of identicals were reported as showing manifest intra-pair differences in severity. In whooping cough such differences occurred in 15 pairs of fraternals and in only 5 pairs of identicals. For all the communicable diseases taken together, 6.8 ± 1.2 per cent of the identicals and 15.9 ± 1.7 per cent of the fraternals gave evidence of intra-pair differences in severity. For the 20 "undetermined" pairs, a much higher percentage of differences was found ($32.7 \pm 4.4\%$). This may be due merely to fluctuation in a small sample, but combined with other findings the data suggest that this is a peculiar group of twins. It is hoped that further study will throw more light on their peculiarities.

2. Operations and accidents

Thirty-six per cent of the individual twins had had their tonsils removed; nine per cent had undergone "other operations." The data for the different groups are tabulated in Table 7. Twice as many fraternals as identicals showed intra-pair differences in incidence of tonsillectomy. Like the contagious diseases, tonsil operations tend to run in pairs, particularly among the identical twins. It is possible, however, that this reflects to some extent the influence of a twin similarity in other traits upon the judgment of physicians and parents. The apparent need for a tonsillectomy in both members of the pair might be emphasized by the thought, "Well, they are alike in everything else, so why not in this?" "Other operations" included those for appendicitis, hernia, sinus trouble, etc. In one case both members of an identical pair of boy twins had a mastoid operation at the same time; one fraternal pair of girl twins had each an "enlarged gland on the neck" removed at the same time—as did one pair of "undetermined" males. The accidents included broken bones, flesh wounds, burns, falls, etc. Apparently, the incidence of operations and accidents was largely a matter of chance,

[8] Siemens (2) says: "But it happens, of course, that one twin may suffer from the after-effects of a disease (that both have had) while his mate does not. I have observed one case of twins in which one had *otitis media* following measles, while the other was free; in another case one twin suffered from paralysis of the eye muscles after they had had diphtheria together."

and rarely occurred to both twins at the same time. The higher percentage of both operations and accidents among the identicals was presumably due to their greater average age.

TABLE 7

Incidence of Operations and Accidents, by Pairs

	NUMBER OF PAIRS IN WHICH BOTH WERE AFFECTED			NUMBER OF PAIRS SHOWING INTRA-PAIR DIFFERENCES IN INCIDENCE		
	Id.	*Fra.*	*Undet.*	*Id.*	*Fra.*	*Undet.*
Tonsillectomy	27	21	4	4	8	1
Other operations	2	1	1	11	6	7
Serious accidents	0	0	0	28	16	7

3. *General health data*

As a further approach to a developmental record of the twins, the mothers were asked about certain characteristics of the general health of each child. The following quotation from the directions for the use of the home interview blanks gives the method used to secure information on the item, "general health." Data on the other traits were secured in the same way:

> The interviewer says, "Now again I am going to ask you to compare the twins in a number of other particulars. If they are practically the same in a certain trait just say 'same,' but if they are different indicate which one is near the average and which one shows the greater difference from the average. . . . . How would you compare them in general health?" Often the mother will state the comparison in somewhat roundabout terms and it is necessary for the interviewer to summarize her statement briefly. In each case, however, it should be possible to read from the blanks whether or not the twins are estimated as being the same or different in general health, and if they are different which one has the poorer health.

The results of this inquiry are tabulated in Table 8.

The figures for the identical and fraternal groups showed varying differences depending upon the trait considered. In "eye trouble" the two groups have about equal numbers showing intra-pair differences. In every other case, the fraternals show the higher percentage; the greatest difference between the two groups being in "health, first year." The differences between the percentages for the two groups, for "health, first year," "susceptibility to colds," and "indigestion" are statistically reliable (4 times the probable error); this is not true of the others, but the fact that the difference always lies in the same direction (except as noted) adds to their significance. The similarity of the two types for the data on "eye trouble" is rather surprising. Visual efficiency is usu-

TABLE 8

General Health Record of Twins

Percentage of Pairs Showing Intra-pair Differences

TRAIT	IDENTICALS	FRATERNALS	* UNDETERMINED	DIFFERENCE BETWEEN ID. AND FRA.
General health	26.0 ± 3.5	29.6 ± 3.7	26.3	3.6 ± 5.1
Health—first year	5.8 ± 2.0	26.1 ± 3.6	18.8	20.3 ± 4.1
Susceptibility to colds	22.7 ± 3.5	39.7 ± 4.0	27.8	17.4 ± 4.2
Indigestion	6.7 ± 2.2	24.2 ± 3.6	23.5	17.5 ± 4.1
Constipation	9.1 ± 2.6	21.5 ± 3.5	0.0	12.4 ± 4.5
Headaches	20.7 ± 3.6	26.6 ± 3.8	31.3	5.9 ± 5.2
Eye trouble	19.4 ± 3.4	19.2 ± 3.3	22.0	—0.2 ± 4.5
Combined traits	16.9 ± 1.2	26.7 ± 1.4	19.8	9.8 ± 1.8

* The number of pairs was too small to warrant computing reliabilities.

ally thought to be largely dependent upon hereditary factors, yet apparently as many identical as fraternal pairs showed differences in this trait. Also a greater similarity might be expected between "general health" and "health, first year."

Of course, all of these traits are measured subjectively and the error of measurement is necessarily large. This factor would vary with the trait measure; probably the mothers' opinion of "health, first year" is the most reliable of all the traits listed. She generally knows if she had much difficulty bringing a child through this period; differences between two children are fairly objective and are well remembered. On the other hand "general health" is such a vague term and applies to such a long period of time that many errors of fact and judgment are probable.

It is also possible that errors due to the subjectivity of the measurement may not equally affect the answers of the mothers of the different types of twins. Mothers of fraternal twins may tend to exaggerate differences or *vice versa*. The problems involved are complex. However, even though they should prove to be an unreliable measure of health conditions they still have significance as a measure of the mothers' attitudes towards twins. It is noteworthy that in general the data are corroborative of the findings from the more objective records of disease incidence; namely, that in traits of health and disease, as well as in various other physical and psychological traits which investigators have studied, identical twins are appreciably more similar than fraternal, or dizygotic twins.

4. Birth weight and weight at the time of the interview

When the members of pairs of twins are measured for any characteristic there is always, of course, a certain amount of difference. In any given case, it would be extremely interesting to know when this difference developed, or what changes have occurred in its magnitude. Measurements taken periodically during the growth of the children would give us this information. In the absence of such cumulative data, the best we can now do is to make use of case reports supplied by persons who have known the twins over a considerable period.

At each interview the mother was asked for the weight of each twin. Often she did not know exactly, sometimes she said that both "always weighed about the same." As the weight at birth had also been secured (4), a comparison could be made between the two. The result of this comparison is summarized in Table 9. There were 123 cases in which an intra-pair difference in birth weights was reported. In 73 of these the direction of the difference was still the same at the time of the interview, in 7 cases the weights had become equal and in 43 cases the direction of the difference was reversed. While such data may be open to question as to the accuracy of the absolute figures, it would appear legitimate to consider the *relative* status of identicals and fraternals for the traits reported on.

TABLE 9

Weights at Birth Compared with Weights at the Time of the Interview

GROUP	NUMBER OF PAIRS WITH DATA AT BOTH PERIODS	PAIRS WITH A "DIFFERENCE" IN WEIGHT AT BIRTH	PAIRS WITH "DIFFERENCE" IN WEIGHT WHEN INTERVIEWED	PAIRS WITH "DIFFERENCE" AT BOTH PERIODS	CASES IN WHICH THE "DIFFERENCE" LAY IN THE SAME DIRECTION
Identical	57	52	43	38	25
Fraternal	65	59	60	54	39
Undetermined	14	12	13	12	9
Totals	136	123	116	104	73

5. Health the first year of life and health at the time of the interview

A similar comparison was made of the ratings on health. A common belief among laymen is that "one twin is usually sickly." Our data, although inconclusive, show nothing to support such a conclusion (Table 10). In 4 cases out of the 9 showing differences at both

periods, the inferior retained his position (in 5 cases out of 9 the position had been reversed); while of the 25 children who had inferior health their first year 21 had in this respect equalled or surpassed their mates when the interview was made.[1]

TABLE 10

General Health the First Year of Life Compared with General Health at the Time of the Interviews

GROUP	NUMBER OF PAIRS WITH DATA AT BOTH PERIODS	PAIRS WITH A "DIFFERENCE" IN HEALTH THE FIRST YEAR	PAIRS WITH A "DIFFERENCE" IN GENERAL HEALTH, TIME OF INTERVIEW	PAIRS WITH "DIFFERENCE" BOTH PERIODS	CASES IN WHICH "DIFFERENCE" LAY IN SAME DIRECTION
Identical	69	4	19	2	0
Fraternal	69	18	20	5	2
Undetermined	16	3	6	2	2
Totals	154	25	55	9	4

A fairly impressive finding is the markedly larger proportion of fraternals showing a difference in "health the first year." The table indicates that such a difference is not important with reference to later health records. It could, however, be important in creating behavior differences.

C. CONCLUSIONS

When the developmental records of identical and fraternal twins are compared it is found that the fraternal show more intra-pair differences than the identical in most traits of general health; they also show more intra-pair differences in the incidence and severity of most of the children's diseases. Although the effect of heredity is not established in quantitative terms, there is strong indication that it plays an important part in these phenomena.

[1] There is a relatively high infant mortality rate among twins (4). Since our sample contains only pairs in which both members had survived until school age it represents a selected group. Pairs with one very weak member would tend to be eliminated before reaching the age of the group we studied. Our sample would be made up of individuals who approached the normal at birth and during early infancy. If a random sample were made of twins one month old the average intra-pair differences in health would presumably be greater than that reported for our sample of school twins. The effect of such selective factors on the sample should be borne in mind when interpreting the data for weight as well as health differences.

Intra-pair differences in general health (as estimated by the mothers) are about equal in the two types of twins at the time of the interview; during the first year of life, however, such differences are much more numerous among the fraternals.

In the combined experience of the mothers, a discriminable health difference during infancy bears no relationship to discriminable health differences during the school period.

A weight superiority shown in one member of a twin pair at birth, tends to be retained into the school period.

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THE BLOOD GROUPS OF THE MAORI

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Recent observations [1,2] of the iso-haem-agglutination reactions of the bloods of pure blood Australian Aborigines have led me to undertake similar work on the full blood Maori of New Zealand.

I approach the publication of this work with the realization that it is but an insignificant step towards the scientific understanding of their blood group relations but with the hope that the observations contained in it will be a stimulus that will produce a carefully planned investigation in the future.

A grant for carrying out the investigation was made by the Australian National Research Council and I was directed by letter to the Honorable Sir Apirana Ngata, Minister for Native Affairs in the New Zealand Parliament. This gentleman's ready appreciation of the difficulties of the task, his enthusiasm for its immediate prosecution and the active coöperation which he manifested by presenting men who occupied high official and social positions in the Maori community to assist me paved the way for this work and resulted in the collection of blood samples from two hundred full blood subjects living in widely separated districts of New Zealand; all in the short time of five weeks.

Whilst under modern conditions the kinship bond is so strong a factor in Maori life that most members of a tribe tend to seek their livelihood within the original tribal boundaries, nevertheless it has not restrained men of special ability in adapting themselves to a new civilization, from taking up responsible positions in cities and districts far from their birthplace. This dispersion has not been inconsiderable and would make necessary a wide search to seek out all full-blood members of a single tribe.

Another time-consuming factor was the social form to be observed by one presented as a guest to the Maori. One must be presented by a man high in the confidence of each tribe, the object of the investigation must be made clear to each individual from whom a blood specimen is

expected and all this done through the medium of an interpreter, as the Maori tongue is generally used and forms a strong bond for the unity of the race.

Again the genealogy of each subject had to be recorded and the purity of his descent confirmed by men well versed in tribal history. It was indeed fortunate that men who were competent in these matters were able to devote so much of their time in planning my journeys, selecting and presenting me to the subjects, recording the genealogies of each one and even assisting in the collection of blood. Kingi Tahiwi of the Native Department in Wellington proved a most able organizer in arranging the venue and directing the work in the south of the North Island of New Zealand. P. H. Tomoana of Hawkes Bay made possible the investigation of members of the Ngati Kahungunu tribe, scattered as they are over many miles of rich agricultural and pastoral lands. In Rotorua Tai Mitchell's interest in the work enabled me to classify one hundred and twenty-seven members of Te Arawa tribe. His graciousness towards me during my stay in Rotorua has made that brief sojourn among his people a delightful memory of a most sincere hospitality.

I quote Raymond Firth [3] when I say that one finds among the Maori a society which has achieved a kind of simple equilibrium not incompatible with a widespread sense of personal dignity and of collective satisfaction.

METHODS

In a previous paper [1] I suggested that in investigating the iso-haem-agglutination reactions of a pure race the scientific method of approach was by cross-agglutination of all sera and corpuscles of large groups of individuals. By this method all fallacies are obviated and a true picture of the agglutinin-agglutinogen systems is obtained.

In another publication,[2] however, I came to the conclusion that it was justifiable to classify the blood of each member of a pure race into one of the four groups of Moss by means of known sera, recognizing, however, that such a method takes no regard of agglutinizable substances other than the two recognized agglutinogens "A" and "B." The two fallacies of such a method therefore are that unknown iso-agglutination systems may be completely overlooked and that anomalous results may be produced by the presence of hetero-agglutinogens in the bloods under investigation.

Three factors conspired against my attacking the problem by cross-agglutination of sera and corpuscles. These were the short working

time at my disposal, the doubling of expense by taking a trained assistant and the then unknown attitude of the Maori towards venipuncture, which is essential for purposes of cross agglutination. For these reasons I made my objective the observation of the reactions of the bloods of a large series of full blood Maori to known Group 2 and Group 3 sera.

In the actual classification I strictly followed the methods adopted in my previous work. Corpuscles collected from each subject were suspended in a solution of normal saline containing .85 per cent. of Sodium Citrate to prevent coagulation. The strength of each corpuscular suspension was adjusted to approximately 2 per cent. of corpuscles. Within twelve hours after collection these suspensions were tested against known Group 2 and Group 3 sera obtained from white people. The testing was done by the drop method on microscopic slides and the mixtures of corpuscles and sera were stirred at regular intervals with glass rods, microscopic examination of each mixture being made after 30 minutes if by that time no naked eye agglutination had become visible. All observations were made at room temperature.

Moss' classification of the blood groups in man is as follows and is used throughout this paper.

	Group 1	*Group 2*	*Group 3*	*Group 4*
Agglutinin	o	b	a	a + b
Agglutinogen	A + B	A	B	O

The results of the classification of two hundred subjects by this method are shown in the following tabulation.

Tribe	*Group 1*	*Group 2*	*Group 3*	*Group 4*	*Totals*
Ngati Kahungunu	0	14	1	25	40
Ngai Tahu	0	1	0	1	2
Ngati Raukawa	0	7	0	17	24
Ngati Tuwharetoa	0	2	0	0	2
Ngati Tuhoreke	0	1	0	0	1
Tuhoe	0	1	0	1	2
Tainui	0	0	0	2	2
Te Arawa	24	53	1	49	127
Totals	24	79	2	95	200

The names, genealogies and respective blood groups of each subject were recorded and are now in the care of Professor A. R. Radcliffe-Brown in the Department of Anthropology at Sydney University.

The present tribal disposition of the subjects classified by me may be recognized in the following map. Comparison of this with the map

published by Raymond Firth showing the original tribal disposition makes obvious the dispersion which has been brought about by modern conditions of life.

DISCUSSION

The most striking feature of these results is that whilst only two subjects out of two hundred belonged to Group 3, twenty-four belonged to Group 1. Now the "A-a" and "B-b" agglutination systems are inherited independently and the percentage of Group 1 is regulated by the fortuitous inheritance of the two systems by one individual. Consequently it is impossible for a race containing 1 per cent. of Group 3 to contain at the same time 12 per cent. of Group 1. The two subjects found to belong to Group 3 were rather isolated types, one being a woman of forty-four years of age, the other a man aged twenty-four. None of their relatives or descendants were available for classification. Although these two people were represented as full blood Maori, it is quite probable, in view of the extraordinarily low percentage of Group 3, that there had been in their case some white intermixture in a past generation. If this is the case, and I think it is the true explanation, then the full blood Maori has no "B-b" agglutination system in his blood. Why then have the corpuscles of twenty-four members of the Te Arawa tribe become agglutinated by the agglutinin "b" contained in the known Group 2 serum? The explanation of this is in my opinion intimately bound up with the early history of the Maori in New Zealand.

The Maori immigrated to New Zealand from Hawaii some six hundred years ago to find an aboriginal Maoriri race in possession of the islands. These Maoriri were characterized by their fair skins and a certain reddish tint of their hair. Many were slain by the Maori immigrants and the remainder driven from the fertile districts to the security of more mountainous country. In later generations a considerable Maori-Maoriri miscegenation occurred until now the Maoriri has disappeared as a race, leaving behind individuals who in many cases still carry in their red tinted hair and fairer skins the evidence of their Maoriri forebears.

I was led to the conclusion that all the subjects apparently belonging to Group 1 were in reality the descendants of Maori-Maoriri unions, by the fact that four of them had fair skins and red tinted hair and were acknowledged by other Maori to have been descended from Maoriri stock. This conclusion was strengthened by the fact that all these individuals were members of Te Arawa tribe, which made its home centuries ago in the Rotorua district on the fringes of the Urawera Mountains, into which the Maoriri retreated. One would suspect therefore from their propinquity alone that Maoriri inter-marriage with Te Arawa tribe would not be uncommon.

It would seem therefore that the explanation of the apparent high percentage of Group 1 among Te Arawa is that in the bloods of certain members of the tribe there is either a new agglutination system, or some new non-specific agglutinogen which has reacted with the agglutinin "b" of the known Group 2 serum and that this agglutination system or agglutinogen has been inherited from Maoriri ancestors.

The weight of evidence therefore seems to point to the fact that the descendants of the Maori immigrants from Hawaii fall into two blood groups, corresponding to Groups 2 and 4 of Moss when classified by means of known sera. This can only be confirmed by cross agglutination of the corpuscles and sera taken from several series of subjects after exclusion of white or Maoriri inter-mixture and the presence of a new agglutination system in the Maoriri strain proved or disproved by the same method.

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My thanks are due to Dr. Lynch of the Wellington General Hospital for the Group III serum with which he provided me.

CONCLUSIONS

I. The pure Maori race probably does not contain the "B-b" agglutination system.

II. Classification by known sera reveals the "A-a" system distributed in two groups corresponding to Groups 2 and 4 of Moss.

III. Further investigations should be based on cross-agglutination methods, care being taken to exclude either white or Maoriri strains.

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NOTES

EUGENICS RESEARCH SURVEY

A critical survey of data relevant to eugenics research is now being prepared under the auspices of the Eugenics Research Association. Mr. Frederick Osborn, Treasurer of the Association, is chairman of the committee in charge of this project. The publication date is tentatively set as January to March, 1932.

In the Eugenics Research Survey special attention will be given to the data bearing upon the operation of social factors affecting the differential natural increase of population groups under different social conditions (demogenics). Dr. Frank Lorimer is in charge of this section of the survey.

The Association will be grateful for the receipt of notices of new research, completed or in process, having possible bearing upon problems of eugenics research, especially in its social aspects. Communications may be addressed to Eugenics Research Survey, American Museum of Natural History, West 77th Street, New York City.

FRANK LORIMER

ERRATUM

My attention has just been called to the fact that in Table 4 of my paper "Biological Factors in Negro Mortality" (HUMAN BIOLOGY, Vol. I, No. 2, p. 240) the headings "Colored males" and "White females" in some manner became transposed during the passage of the paper through the press.

RAYMOND PEARL

RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Department of Biology, School of Hygiene and Public Health, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

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